MAY 11 1932

Medical Life

ARCHIVES OF DISEASE IN CHILDHOOD.

EDITED BY

HUGH THURSFIELD, D.M., F.R.C.P., and REGINALD MILLER, M.D., F.R.C.P.

WITH THE HELP OF

H. C. CAMERON, M.D., F.R.C.P.
H. A. T. FAIRBANK, D.S.O., M.S., F.R.C.S.
LEONARD FINDLAY, D.SC., M.D.
A. DINGWALL FORDYCE, M.D., F.R.C.P.EDIN.
C. MAX PAGE, D.S.O., M.S., F.R.C.S.
LEONARD G. PARSONS, M.D., F.R.C.P.
G. F. STILL, M.D., F.R.C.P.
EDITOR OF THE *British Medical Journal*.

Vol. 7. APRIL, 1932. No. 38.

CONTENTS

PAGE

Kala-Azar in Children of North China. By Annie V. Scott, M.D., and P. K. Li, M.B. 59

Further Note on Gastromegaly from Duodenal Ileus. By Reginald Miller, M.D., F.R.C.P., and H. Courtney Gage, L.R.C.P., M.R.C.S. 65

Multiple Cases of Erythema Nodosum in a Class of School Children. By Albin Landau, M.D. 77

The Extent of the Pancreatic Lesions in Diabetic Children. By Henry E. W. Roberton, M.A., M.B., B.Ch. 83

Difficult and Defective Children. By A. Dingwall Fordyce, M.D., F.R.C.P. ... 89

Four Cases of Urinary Obstruction. By A. V. Neale, M.D., M.R.C.P. 97

ISSUED BY THE BRITISH MEDICAL ASSOCIATION.

London: British Medical Association House,

Tavistock Square, W.C.1.

Yearly Subscription (6 numbers), 25/- *Single Number, 4/6*

GENERAL ADVISORY BOARD.

H. T. Ashby (Manchester).
W. R. Bristow (London).
Alan Brown (Toronto).
E. Cautley (London).
Carey Coombs (Bristol).
Glen Davison (Newcastle-on-Tyne).
T. Y. Finlay (Edinburgh).
G. B. Fleming (Glasgow).
Prof. John Fraser (Edinburgh).
Prof. W. E. Gallie (Toronto).
Sir Archibald Garrod, K.C.M.G. (Oxford).
J. F. Gaskell (Cambridge).
G. R. Girdlestone (Oxford).
H. Tyrrell Gray (London).
Robert Hutchison (London).
R. C. Jewesbury (London).
F. S. Langmead (London).
C. P. Lapage (Manchester).
W. L. Maccormac (Balham).
Helen Mayo (N. Adelaide).
T. P. McMurray (Liverpool).
Charles McNeil (Edinburgh).
A. E. Naish (Sheffield).
D. H. Paterson (London).
W. J. Pearson (London).
F. J. Poynton (London).
T. G. Pugh (Carshalton).
F. C. Pybus (Newcastle-on-Tyne).
Sir Humphry Rolleston, Bt. (Cambridge).
J. D. Rolleston (London).
J. C. Spence (Newcastle-on-Tyne).
E. H. M. Stephens (Sydney).
G. Bruton Sweet (Auckland, N.Z.).
C. W. Vining (Leeds).
E. H. Williams (Dunedin).

NOTICE TO SUBSCRIBERS.

Subscriptions are payable to the Financial Manager of the British Medical Association who has charge of all financial matters concerning the ARCHIVES OF DISEASE IN CHILDHOOD. Address: British Medical Association House, Tavistock Square, London, W.C. 1.

NOTICE TO CONTRIBUTORS.

All papers submitted for publication should be sent to Dr. Reginald Miller, 110, Harley Street, W. 1. All other editorial matters should be referred to Dr. Hugh Thursfield, 84, Wimpole Street, W. 1.

Papers sent for publication should be typewritten. Illustrations will usually be inserted in the text itself, and care should be taken that the text is marked to show the desired position of each illustration.

Charts and curves accompanying papers should be carefully drawn, on tracing linen. Any lettering on these drawings should be lightly inserted in pencil.

Contributors will receive one proof in page, but it is assumed that all but verbal corrections have been made in the original manuscript; an allowance at the rate of ten shillings per sheet of sixteen pages is made for alterations in the proof (printer's errors excepted), and contributors will be responsible for any excess.

Contributors will be supplied with reprints of their articles at cost price if application is made when returning proofs. An estimate of costs will be given if desired on application to the Financial Manager of the British Medical Association.

Papers which have been published become the property of the ARCHIVES and permission to republish must be obtained from the Editors.

KALA-AZAR IN CHILDREN OF NORTH CHINA

BY

ANNIE V. SCOTT, M.D., and P. K. LI, M.B.

(From the Cheeloo University Hospital, Tsinan, Shantung, China.)

Our experience with kala-azar among children is limited to that of two provinces of North China, Hopei and Shantung, and covers the years 1921 to 1930 inclusive. The cases admitted to our service entered through our regular clinic in the city, as we have not attempted to handle these cases in the outlying endemic village districts. We recently had in the clinic at one time a case of chronic malarial fever, a left-sided psoas abscess and a case of kala-azar, each coming from a kala-azar region, and the parent, in each case, announced that he had brought the child for treatment of kala-azar.

L. E. Napier[1] has published several detailed accounts of the clinical and laboratory findings of kala-azar as seen in India. His notes often record cases occurring in children, but we are unable to find any investigation of kala-azar, either in India or China, which is confined to the disease as it occurs in children. In discussing the signs and symptoms of kala-azar in India, Napier states that they ' are most characteristic from 4 to 14 years.' If this is true, it seems well to review the records of a number of children suffering from this disease, and to attempt to formulate a clinical picture of the child affected with it. With this purpose in view, we have reviewed the histories of one hundred children treated on our service. Fifty of these cases were treated between 1924 and 1927, and fifty between 1928 and 1930. In both groups consecutive cases were taken, in order to avoid the possibility of selecting any outstanding symptoms.

By recording a few typical case histories and the physical and laboratory findings in a number of cases of kala-azar occurring in children of North China, we hope to furnish information which may be of assistance to physicians caring for children suffering from this disease.

Case reports.

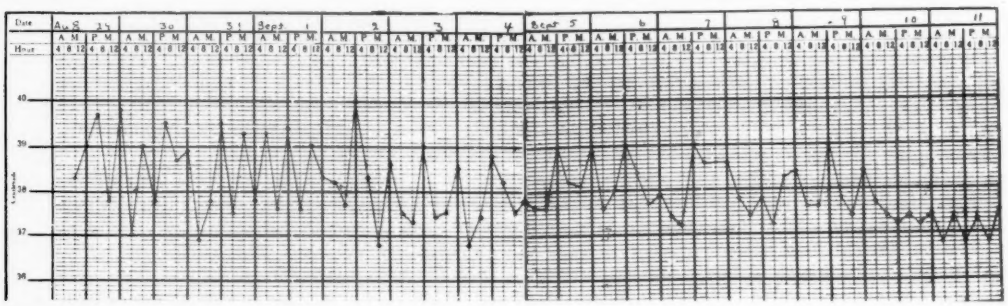
Case 1.—R. W., an American child, female, 2 years of age, became ill February 3rd, 1922. The chief complaints were fever and general malaise. Daily examinations showed no remarkable physical signs. As there was a family history of pulmonary tuberculosis, a careful search was made for the disease. Pirquet's test was negative. X-ray examination of the chest showed no changes of importance. During the second week of illness the patient had a daily afternoon elevation of temperature not exceeding 102°. From February 10th to 28th the temperature was irregular, ranging from 101° in the morning to 105° in the afternoon. During this period the patient's appetite was good. She slept well, played normally, and lost very little weight.

On March 16th the spleen was found enlarged 3 cm. below the left costal margin in the nipple line. The liver extended one finger-breadth below the right costal margin in the nipple line. Blood examination, March 16th, showed red blood cells 4,875,000; Hb. 70 per cent.; white cells 7,150; (polymorphonuclears 35 per cent., lymphocytes 35 per cent., large mononuclears 30 per cent.). On March 18th, Leishmania donovani bodies were found in films made from material aspirated from the spleen.

Some physicians record as a finding characteristic of kala-azar a double rise of temperature within twenty-four hours. Among 120 cases where four-hourly records were kept, only one child showed this double rise.

Case 2.—Li Yu Shan, male, 4½ years of age, was admitted to hospital with the complaint of fever for one month. This fever was reported as being highest in the afternoon (see Chart). During this febrile period, a mass had appeared below the left costal margin and was gradually increasing in size.

Physical examination showed a slightly anæmic, normally developed child. Nutrition was good. There were no remarkable findings on physical examination, except that the spleen was enlarged 7 cm. below the costal margin in the left nipple line. The liver was not enlarged. Films made from material aspirated from the spleen showed Leishmania donovani bodies.



Case 3.—Kiang Nai Hsun, male, aged 7 years and 10 months, entered hospital November 17th, 1930. The chief symptoms which brought the patient to the hospital were enlargement of the abdomen, masses in the upper abdomen, loss of appetite, daily fever and frequent epistaxis. The illness began in May, 1927, with fever and loss of appetite. The parents reported that, after one month of fever, the patient became very emaciated and a mass was palpated below the left costal margin. This mass gradually increased in size and epistaxis occurred frequently. After the fever had continued for two months, a mass was first palpated beneath the right costal margin. The parents reported that the patient had suffered from continuous fever from the onset of the illness.

This is a typical history of kala-azar children coming to us from the villages of Shantung.

Analysis of 100 cases in children.

Sex.—According to information secured from parents coming from kala-azar regions, there is no reason to believe that male children contract the disease more frequently than females. The larger number of males in our figures in children (Table 1) is the same as that seen in our total kala-azar cases. These figures merely mean that some Chinese parents feel it is more necessary to try to preserve the lives of their sons than of their daughters.

Age.—A large number of our cases come from country districts, necessitating several days' travel in order to reach us. These villagers frequently tell us that, in their home village, there are infants only a few months of age apparently suffering from the same disease, but prevented from coming for treatment because of the difficulties of travel. The youngest infant we have seen in the clinic suffering from kala-azar was eight months of age.

TABLE 1.

SEX AND AGE INCIDENCES IN 100 CASES.

Sex.	No. Cases.	Age.		
		Eldest.	Youngest.	Average.
Male	79	12 years	1 year 6 months	7 years 1 month
Female	21	11 years	3 years 4 months	7 years 10 months

History.—The incidence of the disease in the patient's family and among neighbours, the type of onset and the history of symptoms are as reliable as such data can be when collected from parents, relatives or neighbours of the patient.

It is certainly true that persons coming from an endemic region are alert to notice the early symptoms of the disease. It is difficult to secure definite information regarding the course of temperature. Many cases give a history of irregular temperature which is also intermittent. Several come saying that, at the onset of the disease, they had a period of malarial fever. One thing true of all cases is that they run the irregular fever for a few weeks to a few months, before the spleen is enlarged below the costal margin. We have been surprised at the lack of history of loss of strength. Many of these little patients come from farm duties requiring physical endurance. It is remarkable to see these anæmic, emaciated children enter the clinic, having walked fifty or sixty miles in two or three days. The state of nutrition must be the chief factor in causing the gingivitis which affords an excellent nidus for Vincent's spirochætal infection, which is one of the most serious complications met in kala-azar children.

TABLE 2.

CLINICAL HISTORIES IN 100 CASES.

Duration of disease in months.			History of disease same as patient.		Symptons.				
Max.	Min.	Average.	Family.	Village.	Temp. irreg	Diarrhœa.	Epistaxis.	Bleeding from gums.	Loss of strength
42	1	11 months 24 days	17	54	93	20	35	27	23

Physical findings at time of admission.—There are given in the list below the measurements of the spleen and liver taken in the nipple line from the costal margin to the lower border of the organ.

Enlargement of spleen	max.	25 cm.
	min.	3 ,,
	average	10·9 ,,
Enlargement of liver	max.	10 cm.
	min.	2 ,,
	average	4 ,,
Cancrum oris		6 cases.
Œdema (feet)		7 ,,
Heart, functional murmur		55 ,,
Lungs, findings important		0 ,,
Otitis media		4 ,,
Congested superficial abdominal veins		8 ,,
Pigmentation of skin increased		17 ,,

In practically all cases of cancrum oris, the process starts at the gum margin and occurs most frequently in cases with spirochætal infection of the gums. Noma of the cheek, when found, occurs in cases with low red blood counts and suffering from marked malnutrition. Œdema of the feet and legs also occurs with very poor nutrition. It is interesting to see the localized systolic heart murmur disappear as the child's blood-picture and general nutrition improve As most of our patients come from villages where the young boys wear little clothing, it is very difficult to judge as to pigmentation of the skin. The pigmentation which we have recorded is dark brown, distributed over the lateral sides of face, external surfaces of forearms, dorsum of the hands, dorsum of the feet and extensor surfaces of the legs.

Enlargement of the spleen is a constant sign in patients coming to us with kala-azar. Case No. 1, which was under our care from the onset of illness, showed a spleen 3 cm. below the left costal margin six weeks after the onset of the symptoms. Most parents say that the mass was first palpated below the left costal margin, one to three months after the child began to run irregular fever. There is a constant relation between the period of illness and the size of the spleen. Many cases show no enlargement of the liver, even when the spleen extends well into the left lower quadrant of the abdomen. Malnutrition is not listed in the above findings, for every child coming to us with kala-azar shows malnutrition. More often than loss of strength loss of weight is given as one of the progressing clinical symptoms of kala-azar in children. The child who has been ill with kala-azar for six months is emaciated, looks tired and anæmic, and has a markedly enlarged abdomen. The child who has had kala-azar one year is the typical famine-district child:—peaked features, tired, disinterested eyes, limbs covered with skin only, swollen feet, protuberant abdomen. His one desire in life is to be fed and allowed to sleep almost continuously.

Laboratory examinations at time of admission.—Stool cultures and smears from the gums were made in certain cases only; the other laboratory results reported were routine work.

Routine examination of stool-smears gave the following results:—ascaris ova, 54 cases; hookworm ova, 10 cases; trichomonas, whipworm, and giardia, 1 case each. On stool culture, dysentery bacilli were found in 3 cases. In the examinations of smears from the gums the spirochæte and fusiform bacilli of Vincent's infection were found in 41 cases.

Later investigations lead us to believe that our figures are low for incidence of intestinal bacillary dysentery infection and for Vincent's spirochætal infection of the gums.

The children showing abnormal urinary findings have normal kidney function, and the abnormal findings in the urine rapidly disappeared as the child's general condition improved under kala-azar treatment. The routine urinary analyses showed negative results in 83 cases; and albumin and casts in 16 and 6 cases respectively.

TABLE 3.

BLOOD AND PUNCTURE EXAMINATIONS.

Films made from						Blood examination.			
Liver puncture.			Spleen puncture.						
Leishmania donovani found.	Leishmania donovani not found.	No puncture.	Leishmania donovani found.	Leishmania donovani not found.	No puncture.		Max.	Min.	Average.
						R.B.C.	4,960,000	640,000	1,163,000
						Hb. %	80	18	42·5
						W.B.C.	11,000 (Noma)	1,000	4,238
						Poly.	79 %	27 %	52·5 %
						Lymph.	65·5 %	15 %	33·5 %
60	4	31	46	2	49	L.M.	25 %	2 %	9·5 %

The characteristic blood-picture of our kala-azar cases shows a low total white count and an increase of large mononuclear cells. The hæmatocytic change is that found in marked secondary anæmia.

We have found that films made from material aspirated from the spleen afford the most rapid and accurate diagnosis of kala-azar. Even in cases with prolonged bleeding time we feel it is safe to do a spleen puncture. If it

is possible (where the bleeding time is prolonged) we transfuse before the initial spleen puncture.

Practically all our patients suffering from kala-azar show a positive serum globulin test[2] which persists, following treatment, even after the spleen films are negative for Leishmania donovani bodies. We feel warranted in doing a spleen puncture to search for Leishmania donovani bodies, if a patient shows positive serum-globulin test. This test consists in adding a small quantity of blood, obtained by skin puncture, to 10 to 30 times its volume of distilled water contained in a small test tube. In kala-azar the solution becomes turbid at once, and, on standing, a heavy, white precipitate is formed. (Our routine is blood 29 c.mm., distilled water 0·6 c.cm.)

Summary.

By means of case reports and tabulations of clinical and laboratory findings, we have attempted to give a picture of the child of North China suffering from kala-azar. He is distinctly a child suffering from a chronic devitalizing malady. He comes from a village where the disease is endemic, or has recently resided in such a district. He has suffered from irregular fever and progressive loss of weight since the onset of the illness. The child is emaciated, looks anæmic and always has enlargement of the abdomen. Secondary anæmia, with a low total white count and increase of the large monocytes are the characteristic laboratory findings. The enlargement of the spleen is definitely related to the period of illness. Heart and kidney signs when present are not serious and clear up under treatment for kala-azar.

REFERENCES.

1. Napier, L. E., *Ind. Med. Gaz.*, Calcutta, 1921, LVI, 401.
2. Hsieh, H. P., and Hsien Wu, *China Med. J.*, 1921, XXXV, 527.

EDITORS' NOTE.—It is hoped to publish later a further paper by the authors dealing with the treatment of Kala-azar.

FURTHER NOTE ON GASTROMEGALY FROM DUODENAL ILEUS

BY

REGINALD MILLER, M.D., F.R.C.P.,
Physician, St. Mary's and Paddington Green Children's Hospitals, London,

and

H. COURTNEY GAGE, L.R.C.P., M.R.C.S.,
Radiologist, St. Mary's Hospital, London.

Early in 1930 two cases were shown under the title of ' gastromegaly ' by one of us (R.M.) before the Section for the Study of Diseases of Children of the Royal Society of Medicine[1, 2]. In the same year we together recorded in this journal[3] nine cases of the same type under the title of ' Gastromegaly and Chronic Duodenal Ileus in children.' The word ' gastromegaly ' was used to connote an enlarged and hypertrophied stomach which is the most striking feature in these children, and in our paper we claimed that the abnormality of the stomach was secondary to some degree of obstruction in the latter part of the duodenum where this passes under the root of the mesentery and (usually) the superior mesenteric artery (chronic duodenal ileus or arterio-mesenteric compression). In the two years that have passed since our paper appeared, although we have had the pleasure of seeing three cases with medical colleagues and friends, so far as we know no instance of the condition has been exhibited or recorded. From this we infer that the evidence we brought forward of the presence of an obstructive factor in these cases, and more particularly of its site and nature, has failed to carry conviction to others. Nor does this surprise us: we recall that we had great difficulty in recognizing the obstructive element in our early cases, and that it was only by operation on two cases in our series that its position and nature were disclosed.

In the present paper we bring forward two new items of evidence in support of our view that arterio-mesenteric compression is the correct explanation of our cases of gastromegaly. First, we record the case of a baby who died on the ninth day of life with symptoms suggestive of duodenal atresia, and in whom post-mortem examination discovered a definite degree of obstruction of the duodenum as it passed under the root of the mesentery. Secondly, we produce skiagrams taken in the case of a girl aged eight years in which the symptoms, signs and reaction to treatment are the same as in our previously published cases. The skiagrams show the duodenum quite clearly and suggest the presence of obstruction in its third part.

Gastromegaly syndrome.—Before going further it will be well to give as briefly as possible the salient features of gastromegaly cases as described at length in our previous paper.

The most constant early symptom is that of refusal of food rather than vomiting, although in some instances vomiting occurred early enough to raise a suspicion of hypertrophic pyloric stenosis. In its early days the baby feeds extremely slowly,

never welcomes its bottles and usually fails to finish them: only if food is pressed on it does vomiting occur. When it does, the vomiting is forcible in type. Bile may or may not be recognizable in the vomit: it is by no means constant. In the second year, as the child gets old enough to have solids added to its liquid food and to be pressed to take more than it wants, vomiting becomes more troublesome and tends later to occur in attacks accompanied by high fever, much prostration, and often diarrhœa with thick sodden mucus in the stools. The vomit is projected with force, and may show the presence of food taken many hours previously. In addition it contains a great excess of mucus, indicating the presence of gastritis. As a result of the anorexia and the vomiting attacks the child gains weight with extreme slowness, and its physical development is of the frailest though actual stunting in height is not a prominent feature. In between the vomiting attacks the child's appetite remains deplorably bad, its breath foul and its tongue coated. Persistent hiccup may be a conspicuous sympton.

In the minority of cases vomiting is even less conspicuous, and the attacks of fever are associated chiefly with diarrhœa in which the motions contain thick sodden mucus, evidently from the stomach rather than the intestine.

On examination the abdomen is rather protuberant, but it is noticeable that it is chiefly prominent in its upper part. The outline of the enlarged stomach is often to be seen, and on palpation the wall of the stomach gives the sensation of being thickened. After a meal or after inflation of the stomach by a draught of soda-water peristaltic waves may be seen passing from left to right in the region of the stomach. Neither fullness nor peristalsis of the duodenum can be observed, at all events in the absence of vomiting. In addition to these signs splashing in the stomach can be elicited at almost any hour of the day.

X-ray examination shows enlargement of the stomach, the body of the organ often protruding beyond and so obscuring the pyloric end and duodenal cap. In addition hyperperistalsis is well marked, and the emptying time of the stomach much prolonged.

Under treatment by means of a dry diet and an alkaline antiseptic mixture considerable improvement occurs. Vomiting attacks become less frequent or disappear, the tongue cleans, the appetite improves and the hiccups cease to be peculiarly troublesome. The older children do better under treatment than the younger ones since there is probably some tendency for spontaneous improvement to occur at or after the age of six or seven years of age. If treatment is started for the first time at about this age, the improvement obtained may be dramatic (see Case 2 below), since it coincides with the time of a slow spontaneous tendency towards improvement.

Present cases.—The details of the two cases on which we wish to base certain arguments are now given.

Case 1. Barbara S., aged 5 days, was admitted on November 21st, 1931, to Paddington Green Children's Hospital under the care of one of us (R.M.), having been referred by Dr. T. P. Lewis of Braintree, Essex, as a possible case of duodenal atresia.

The child was born at full-term and though small was apparently healthy. From the first she had vomited 'everything,' breast and cow's milk. On two occasions the vomitus had contained black or brownish-black material, the nature of which was unexplained. The vomiting continuing the baby was referred to hospital.

On admission the baby was small and rather dehydrated. On being fed it squirmed about as if in discomfort until relieved by vomiting. Apparently the bulk of the food taken was expelled. The vomit showed no bile or other abnormal colouration. Much mucus was present in it. Irregular gastric peristalsis was observed but it was rather obscured by the perpetual wriggling of the child. A small hard lump was felt far down in the right loin. This was not mistaken for a hypertrophied pylorus, but its nature was not understood. It was probably the tip of one of the lowest ribs left uncovered by the absence of the right kidney as was discovered at autopsy. The motions were dark (meconium).

Vomiting continued. Most feeds were ejected soon after ingestion, though occasionally one would be retained until the next feed. In an effort to see how the stomach emptied an opaque meal was given. This (Fig. 1 and 2) showed that very little food passed into the intestine, though two days later traces of barium were recognized in the stool.

On November 23rd the child was going downhill and in the hope that the obstruction might be due to some condition such as a band that could be dealt with easily, laparotomy was performed. The stomach was found to be large, the pylorus

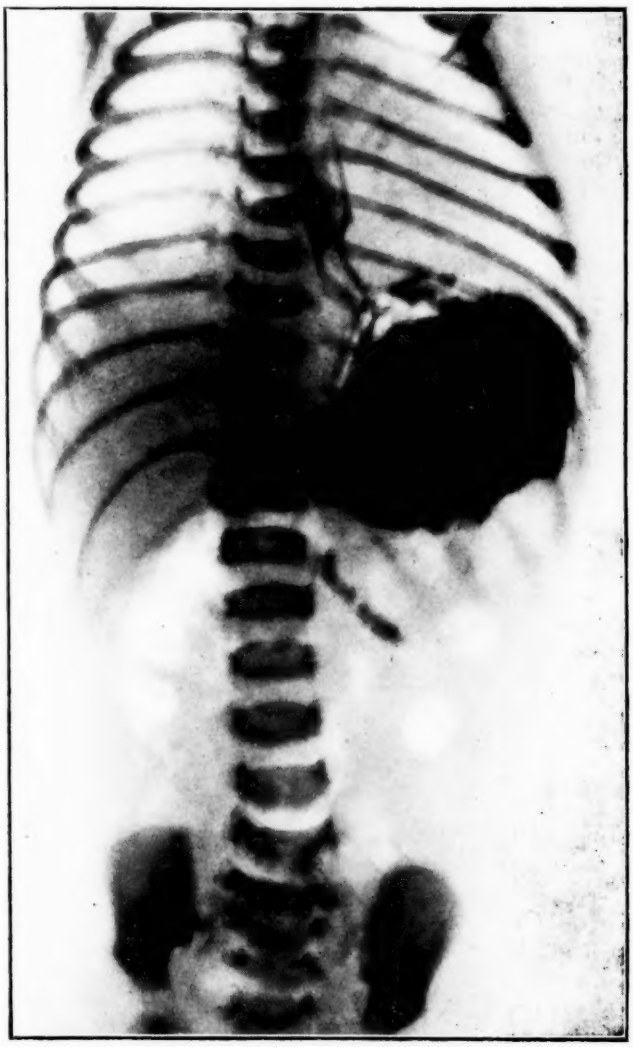


FIG. 1.—Case 1, opaque meal at 15 minutes; prone.

normal, the duodenum large and the jejunum definitely small; but in the few moments available to the surgeon he could not localize the cause of obstruction. The child died next day in hyperpyrexia, aged 8 days.

RADIOGRAPHIC EXAMINATION (November 21st, 1931). By means of a stomach tube $1\frac{1}{2}$ oz. of a fluid suspension of barium sulphate were introduced into the stomach. On screening vigorous gastric peristaltic waves were seen.

The first film (Fig. 1) was taken at 15 minutes in the prone position. It shows a flocculent shadow of the barium, due to admixture in the stomach with the contents present before the barium meal was given. The outline of the shadow is

irregular and does not indicate in silhouette the true contour of the stomach, but only a puddle resting on the anterior gastric wall. It does not reach the fundus, neither does it fill the pyloric antrum as this part passes to the right and backwards towards the duodenal cap. This indicates that the stomach is larger than the patch of barium at first sight suggests. The vigorous peristalsis has already ceased, although only a few minims of the meal have been passed on into the small intestine, to be seen in the commencement of the jejunum as a quite narrow streak. No part of the duodenum is seen at this examination, but it is of interest to note that the streak in the jejunum is at most not more than half the width of the short part of the duodenum seen in Fig. 2.

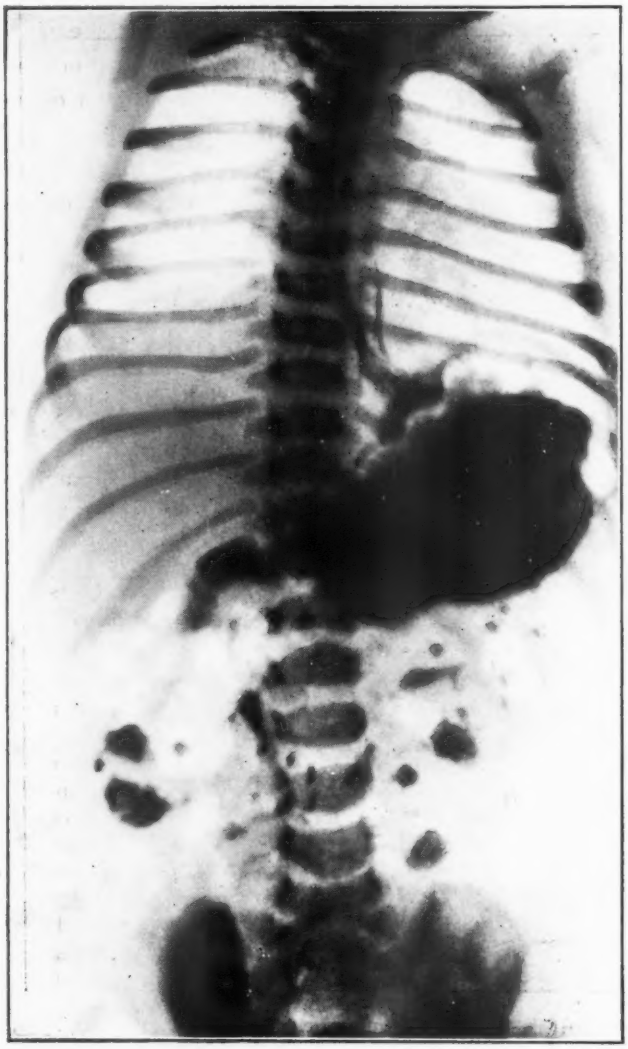


FIG. 2.—Case 1, opaque meal at 35 minutes; prone.

The second film (Fig. 2) was taken at 35 minutes. The general appearance of the stomach remains unchanged and only a few more minims of the meal have passed on, to be seen as flecks distributed throughout the small intestine. This film, however, shows the duodenal cap and a small quantity of the meal in the second or descending part of the duodenum.

No examination was made to determine the exact emptying time of the stomach, the presence of gastric delay being already established. Normally the stomach commences to empty even before ingestion is complete.

AUTOPSY. The body of a very small emaciated baby showing an abdominal wound. On opening the abdomen the stomach was seen to be moderately enlarged and the duodenum, also enlarged, was in proportion to the size of the stomach. Hypertrophy of the gastric wall could not be recognized with any assurance. The jejunum was definitely smaller than the duodenum, between one-half and two-thirds of the width of the duodenum, and the change in the size of the intestine came abruptly at the level of the crossing of the gut by the mesenteric root and the superior mesenteric artery. The right kidney was completely absent: the left enlarged.

Except for the compression exercised on the duodenum the disposition of the root of the mesentery and its vessels appeared normal.

The points in the case related above to which we call special attention are these. In the first place the anatomical picture found post mortem was exactly the same as that found in our two previous cases at operation (Cases 1 and 2 in our former paper). Although clearly the degree of obstruction by arterio-mesenteric compression was more severe than in our previous cases, yet by no means could the present case be classed amongst the examples of congenital megaduodenum in which the duodenum may attain the size of a 'second stomach.' Herein lies a matter of practical importance for in the cases of our group there seems no possibility of performing a duodeno-jejunostomy as may be done to relieve a condition of megaduodenum. Secondly, the radiographic examination, although admittedly undertaken in circumstances of difficulty, proved the presence of obstruction to the evacuation of the stomach but gave no certain evidence as to the site or nature of the obstruction. In this it was exactly in line with the results of our previous cases, though in this fatal case the character of the obstruction was unmistakable post mortem.

Case 2. Betty B., aged 8 years: referred by Dr. W. Horsfall Chesters, of Bromley, Kent, for vomiting attacks accompanied by high fever. First seen July 23rd, 1931.

Child was born at full-term, weighing 7½ lb. From the first there was great difficulty in getting her to take her bottles. She had to have much smaller feeds than an ordinary child as she refused to take more. If pressed she would be sick, the vomit being projected and always containing mucus and sometimes bile. Her gain in weight was very slow, seldom more than 2 oz. a week. At 11 months she refused bottles altogether and they had to be given up. At a year she was very small but plump. She was not regarded as an ill child during her first year: her frailty and peculiar feeding were attributed to the comparatively elderly age of the parents who had been married for seven years before pregnancy occurred.

During the second year great difficulty was experienced in introducing solid food into the diet. Vomiting still occurred if food was pressed. The meals were extremely small ('an eggcupful'), and an hour and a half would be taken to eat a small slice of bread and butter.

In her third year she had whooping-cough very severely and after that the vomiting became worse. Since then she has had bouts of vomiting every 4 to 6 weeks, coming on without warning. The first thing noticed wrong is usually fever, the temperature running up to considerable height, often 105°. Very quickly the vomiting will start and will last perhaps 48 hours, the vomit being full of mucus and often containing bile. The temperature will not regain normal for 4 or 5 days. With the vomiting there is nearly always diarrhœa: the stools contain much thick sodden mucus, 'like skins.' The motions are sometimes putty coloured.

Apart from these attacks the tongue is perpetually coated and the breath foul. Hiccups are frequent. The bowels are very regular: no aperient is ever needed.

Seen in July, 1931, she complained of the same symptoms as above. She was a good height for her age, but very thin and rather underwieght. Her complexion was good. Her tongue was coated. The abdomen was blown out in its upper part, where the outline of a large stomach could be seen. Splashing was easily elicited. Gastric peristalsis was observed.

RADIOGRAPHIC EXAMINATION. A 10-ounce barium meal was given of Allenbury food, milk and chocolate.

The tone of the stomach is exceptionally good, and hyperperistalsis is present throughout the whole of the examination. In spite of this there is an appreciable residue in the stomach at 4 hours and a trace at 8 hours. There is a definite tendency for the pyloric portion to pass well over to the right, but owing to the strong contraction of the pyloric antrum the duodenal cap is not obscured as so frequently happened in the previous series of cases. The duodenal cap is normal, and a normal pyloric canal is to be seen in the first exposure of the serial film, and also on the film taken at 2 hours.

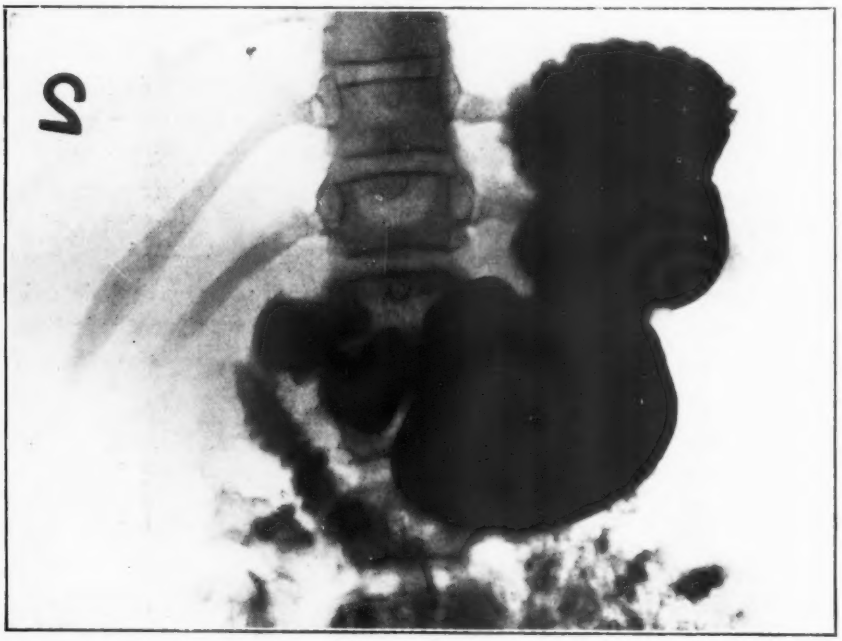


FIG. 3.—Case 2, opaque meal at 2 hours; prone. (See Fig. 5.)

The film at 2 hours (Fig. 3) also shows barium in the descending and commencement of the transverse part of the duodenum, terminating just before the mid-line is reached, and it is interesting to note that it gets wider as this part is reached. There is a definite suggestion of ' cogwheel ' formation, but it is certainly not so square-cut as it is seen to be in adults with duodenal stasis.

Barium is also to be seen in this segment in the 4 hour film (Fig. 4), but unfortunately its termination is obscured by the barium in the transverse colon at the right border of the vertebrae. It certainly does not appear at the left border, and it probably terminates in the mid-line as in the 2-hour film.

There is no ileal stasis, and the passage of the meal through the colon presents no unusual features.

TREATMENT. The girl was treated on the lines described in our previous papers by means of a dry diet, fluids 3 hours or more after meals, and an alkaline antiseptic mixture after food.

COURSE. Seen again in February, 1932, a great improvement was reported. She had gone seven months without being sick, but had then awakened one morning

and vomited bile and mucus, and had passed putty coloured motions. She had gained several pounds in weight. The hiccups and splashing were less, according to the mother. Splashing could, however, easily be elicited. The distension of the stomach appeared less, and the tongue cleaner. The mother stated that if the medicine were left off for three or four days the tongue coated over and the breath became foul. There was still no real appetite: the meals were taken in normal time, but only about a quarter of the normal amount of food was consumed.

In this case we lay stress on the fact that radiographic evidence suggesting obstruction in the latter part of the duodenum was obtained in a typical example of gastromegaly with characteristic symptoms dating from birth in the usual way.

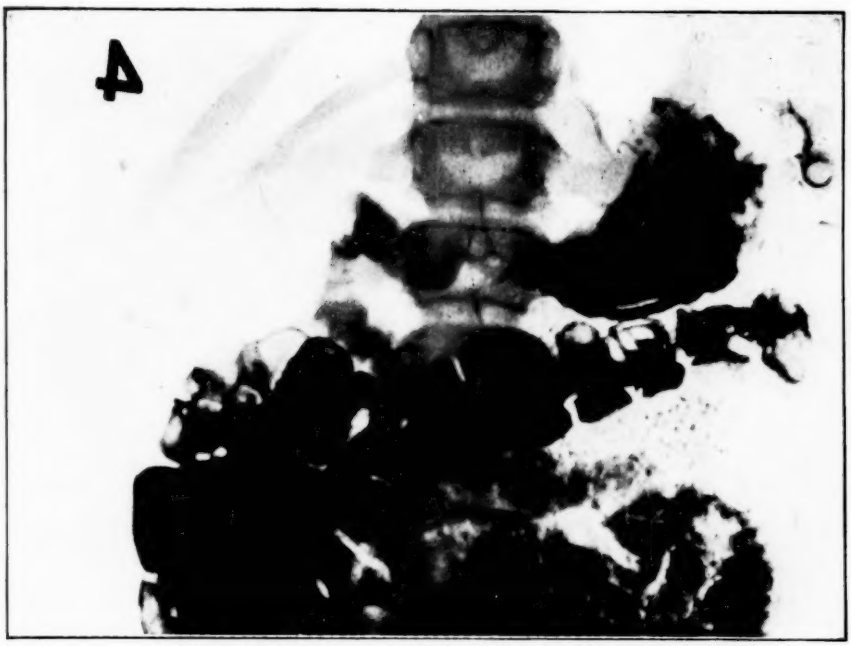


FIG. 4.—Case 2, opaque meal at 4 hours; prone. (See Fig. 5.)

Discussion.

We now proceed to discuss seriatim the points of difficulty in connection with these cases which we have termed 'gastromegaly' and explain as secondary to some degree of obstruction to the duodenum from arterio-mesenteric compression (chronic duodenal ileus).

1. The presence of organic obstruction.—We take first the essential point that there is in our cases some degree of organic obstruction high in the alimentary tract, and that we are not dealing with examples merely of such conditions as pylorospasm, chronic gastritis or atonic dyspepsia. To us the evidence on this point seems quite satisfactory and to be recognized with no great difficulty in practice once its possibility is in mind.

Clinically the stomach may be seen to be enlarged, often causing prominence of the upper part of the abdomen. Hypertrophy of the wall of the stomach is not only suggested by the feel of the organ through the abdominal wall, but is definitely proved by the presence of visible gastric

peristalsis which may be observed after a meal or a drink of soda-water. The large size of the vomits and their projectile character also suggest an enlarged and hypertrophied stomach, such as can only be conceived as secondary to some form of obstruction to its evacuation. Confirmatory evidence of such

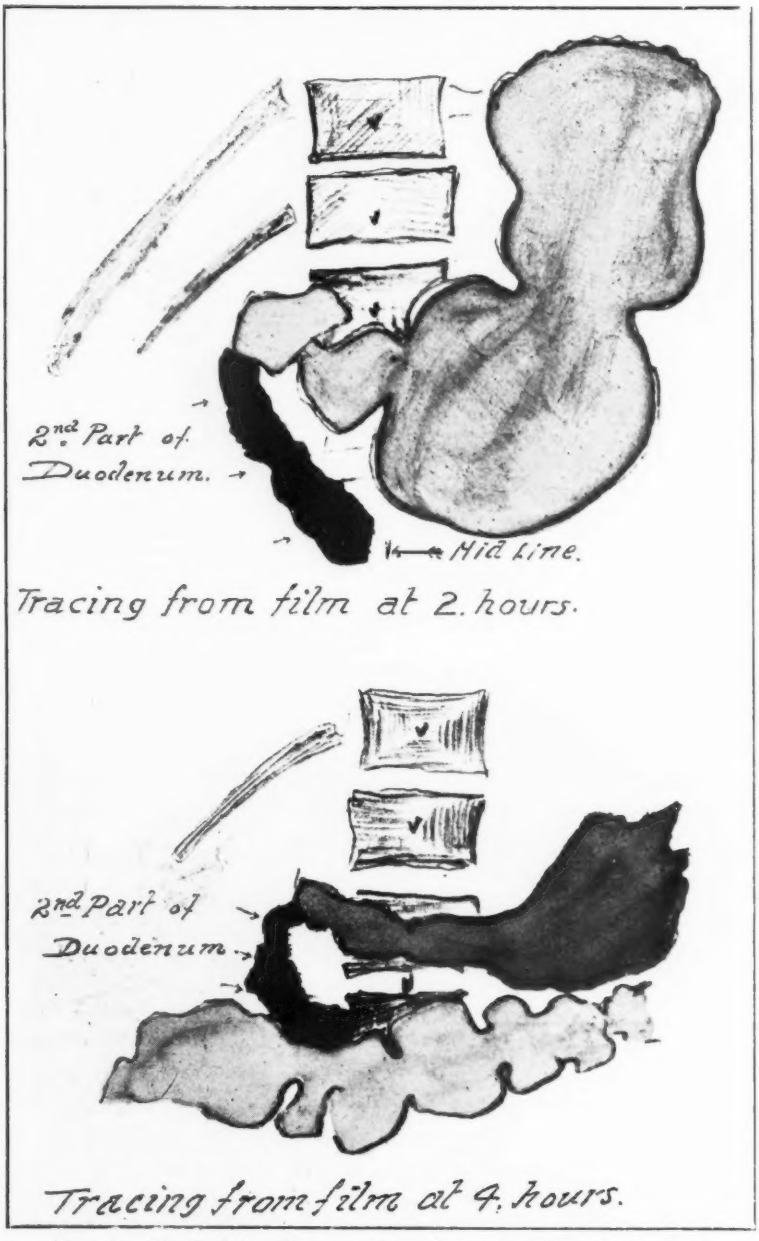


FIG. 5.—Marked tracings of films shown in Figures 3 and 4.

obstruction is found in the fact that, in spite of over-active gastric peristalsis, there is considerable gastric stasis. Clinical proof of this is the presence in the vomit of food taken many hours previously. The perpetual splashing noises which can be heard on shaking the abdomen at almost any time

confirm this. Lastly, the presence of chronic gastritis in a baby or young child does strongly suggest the presence of some high obstruction (cf. the gastritis of hypertrophic pyloric stenosis).

Radiographic evidence of organic obstruction is equally clear. The stomach is abnormally large, often so greatly enlarged is it that the body of the organ covers and obscures the pylorus and duodenal cap. Hyperperistalsis is seen, but in spite of this the emptying time of the stomach is unduly long. In some instances only half the meal may have passed on in $3\frac{1}{2}$ hours. With improvement under treatment the stomach, although still enlarged, becomes smaller and more normal in shape, and the hyperperistalsis may succeed in bringing the emptying time within the normal limit.

Finally, organic obstruction was found in both the cases of our series submitted to operation (Cases 1 and 2 of our previous paper), and in our single fatal case (see Case 1 above).

We conclude, therefore, that in our cases of gastromegaly the presence of some organic obstructive factor is clearly proved, laying particular emphasis on the association of persistent hyperperistalsis with well marked stasis in the enlarged stomachs of these children.

2. The duodenal site of the obstruction.—To prove the presence of obstruction in these cases is far easier than to determine its exact situation, and we must now discuss the evidence for and against our contention that it is in the latter part of the duodenum.

Evidence for duodenal site.—The following arguments may be put forward in favour of this view.

(a) On clinical and radiological grounds we can be sure that the obstruction is in these cases high in the digestive tract, and there is seldom any real difficulty in excluding the stomach itself as the seat of the obstruction by means of the X-ray examination. This is certainly true once experience has been gained of this type of case, and taken by itself it is an important step towards incriminating the duodenum.

(b) Interference with the flow of bile is to be noted in many instances. This does not always take the form of the vomiting of bile, as will be discussed later, but may show itself by gross, though transient, interference with the absorption of fat in the intestine. This is sufficient in degree to make it a wise rule to feed these children with a diet containing a low, rather than a high, proportion of fat.

(c) In our previous paper we stated that the attack of vomiting showed 'nothing sufficiently peculiar to distinguish them as of duodenal origin.' We should now be inclined to modify this statement, and to say that the rapid onset of very high fever, especially if accompanied by the vomiting of bile or the passage of putty-coloured stools, is rather suggestive of the severe toxicosis of duodenal stasis.

(d) Duodenal obstruction was found in the two operation cases previously reported, and in the fatal case recorded here. We also think that the radiographic evidence in Case 2 above is quite satisfactory as proving the duodenal situation of the obstruction. Except that in the fatal case the condition was much more severe than in the others, we are not able to distinguish any difference between these four ' proved ' cases and the rest of our series.

ARGUMENTS AGAINST DUODENAL SITE.—We come now to consider the arguments that can be advanced against the duodenal situation of the obstruction in these cases.

(a) Undoubtedly the chief difficulty in accepting our interpretation of these cases lies in the fact that the opaque meal cannot be relied on to demonstrate the presence of duodenal obstruction. It is certainly true that in nearly all cases, at all events in the absence of vomiting at the time of the examination, the duodenum is not seen. In about twenty cases the only instance in which the duodenal obstruction has shown is that of Case 2 reproduced here.

The question at issue is, therefore, whether these usually negative results preclude the diagnosis of duodenal obstruction. In our view they do not, for several reasons.

In the first place the radiographic results were as negative in our ' proved ' cases as in those unverified by operation or post-mortem examination. Even in our fatal case (Case 1 above), although the obstruction was much more severe than in any of the others, the X-ray examination gave no clear indication of the duodenal obstruction which was so unmistakable post mortem. Secondly, except in the fatal case, we have had no opportunity of examining by radiograms any case during an actual bout of vomiting. Perhaps in such a phase good fortune would lead to a clear picture of duodenal obstruction. Thirdly, our present Case 2, showing a successful radiogram, is exactly in line with the unsuccessful cases as far as clinical history and physical signs are concerned. Lastly, if we grant for the moment that the obstruction is due to duodenal ileus, it is well known that in adult examples of this condition there is considerable difficulty in making a satisfactory diagnosis by means of X-ray examination. A recent paper by R. P. Rowlands[4] has emphasized this once more. The reason for this difficulty will be shown later in discussing the congenital and accessory factors producing the obstruction.

Although, therefore, in our earlier cases we felt considerable hesitation in accepting as true that duodenal obstruction of this type could pass unrecognized in skiagrams, we are now sure that this may be so. In this connection it is to be realized that the enlargement of the duodenum is comparatively slight: in none of our series of cases was there a real megaduodenum.

(b) Another difficulty lies in the fact that the vomiting of bile is by no means necessarily a conspicuous symptom. In the very severe cases of other

authors, associated with megaduodenum, the vomiting of bile has been often a very obvious and suggestive feature, but in our own group, where the obstruction is of much less degree, this is not the case. Even in the fatal instance recorded here, no bile was recognized in the vomits from the fifth to the ninth day when death occurred. There was a history of dark vomits soon after birth. It was suspected that these contained altered blood, but it may have been bile or meconium-stained fluid. In Case 2 above, bile was sometimes vomited apart from food, but in the majority of our cases biliousness of the vomit has not struck the parents as peculiar. Frank[5] has noticed the same thing in cases of duodenal obstruction and suggests that the colour of the bile is lost by dilution.

Whatever the explanation may be we do not regard this difficulty as disproving the presence of duodenal obstruction. In the first place there has been no difference in this point between the cases proved by operation or post-mortem examination and the others; and secondly, there is evidence, as we have already pointed out, that the flow of bile may be interfered with in the fairly common occurrence of pale and fatty stools.

3. Nature of the duodenal obstruction.—If duodenal obstruction be accepted as the explanation of our cases, there is no great difficulty in accepting arterio-mesenteric compression as the cause of the obstruction. In very severe cases giving rise to urgent symptoms in the earliest days of life, as in Case 1 above, it must be, as far as we can see, impossible to exclude duodenal stenosis, obstruction by band or gross malformation of the mesentery; but in the less severe types, with their history of congenital anorexia, vomiting attacks, chronic gastritis and very slow development, it is not easy to think of any condition so likely to be present as that of chronic duodenal ileus such as we found in our ' proved ' cases. As things go the syndrome we have sketched out is a tolerably constant one, sufficiently so to convince us that the same lesion is responsible for all the cases.

Arterio-mesenteric compression has been described as a cause of megaduodenum in a fair number of instances (see Table 1 in our previous paper)*, so that there is nothing unexpected in its causing an obstruction of a milder degree.

There is one other point which to our minds is in favour of chronic duodenal ileus as the cause of the gastromegaly syndrome which we will now discuss.

4. Congenital and accessory factors.—In the production of obstruction by duodenal ileus there are two possible sets of factors. First, there is the strictly congenital factor, the malformation by which the space for the passage of the duodenum is narrowed by the arterio-mesenteric compression. Secondly, there is the accessory factor whereby the space is still further reduced by a drag on the mesentery from a distended stomach or colon.

* An interesting example of megaduodenum of this type which survived until the age of six years and was then successfully dealt with by duodeno-jejunostomy has been recently published by U. Camera[6].

In our cases it seems that the influence of both these factors can be recognized. In our best marked examples there is no doubt some degree of obstruction present from birth. Its severity varies in the different cases but it remains in evidence for many years until, probably by the natural growth of the parts, it ceases to give rise to obstructive symptoms. But it also seems that the accessory factor of mesenteric drag comes into play. The influence of a distended stomach in our cases is quite clear: to press food on the child is to produce vomiting or initiate one of its ' attacks.' The influence of a distended colon is much more difficult to trace, but it is noticeable that in several of our cases the colon has been particularly voluminous and in one instance (Case 9 of our previous paper) was enormous. It is probable that the dual character of the obstruction in these cases accounts for the uncertain radiographic results and the inconstancy of bilious vomiting in them.

From these considerations we submit that the explanation that we have advanced for our cases of gastromegaly, namely, the presence of chronic congenital duodenal ileus, does fit in with the observed facts particularly well. And in this connection we recall that D. P. D. Wilkie[7] found that in many of his adult cases of chronic duodenal ileus there was a history of symptoms during childhood, followed by a period of freedom lasting for many years.

Summary.

Two fresh pieces of evidence are brought forward to support the view published in this journal two years ago, that chronic (congenital) duodenal ileus or arterio-mesenteric compression is the cause of a syndrome in children in which enlargement and hypertrophy of the stomach (gastromegaly) is the most prominent physical sign.

These consist of (1) post-mortem evidence in a baby dying on the ninth day of life; and (2) radiographic evidence in a child of eight years old.

We discuss the arguments against our hypothesis which are based on the practical difficulty of demonstrating duodenal ileus by radiology and other means; and we dismiss them on the grounds that the same difficulties are encountered even in the examples which are verified as correct by operation or post-mortem examination.

We are indebted to Dr. Tierrens, radiologist, and Miss Frost, radiographer, to the Paddington Green Children's Hospital, for the films of the first case recorded here.

REFERENCES.

1. Miller, R., *Proc. R. Soc. Med.*, Lond., 1929-30, XXIII, (*Sect. Study of Dis. Child.*), 33.
2. Miller, R., *Loc. cit.*, 66.
3. Miller, R., & Gage, H. C., *Arch. Dis. Childh.*, Lond., 1930, V, 83.
4. Rowlands, R. P., *Guy's Hosp. Gaz.*, Lond., 1930, XLIV, 93.
5. Frank, E. S., *Ztschr. f. Kinderh.*, Berlin, 1913, IX, 99.
6. Camera, U., *Arch. d. méd. des enf.*, Paris, 1930, XXXIII, 583.
7. Wilkie, D. P. D., *Brit. J. Surg.*, Bristol, 1921, IX, 204; *Brit. Med. J.*, Lond., 1922, ii, 1219; *Amer. J. Med. Sci.*, Philad., 1927, CLXXIII, 643.

MULTIPLE CASES OF ERYTHEMA NODOSUM IN A CLASS OF SCHOOL CHILDREN

BY

ALBIN LANDAU, M.D.

(From the Children's Hospital, Gothenburg, Sweden.)

It is widely, though not universally, accepted that most cases of erythema nodosum in children are connected with tuberculous infection. The frequency of positive tuberculin reactions in children with erythema nodosum almost reaches one hundred per cent. Patients who do not react positively to tuberculin, even in high doses, are insignificant in number: in Dr. Wallgren's statistics here about two per cent.

The characteristic moment for the appearance of erythema nodosum in tuberculous infections is the end of the incubation period, when the organism is in an allergic condition; erythema nodosum is a parallergic phenomenon. This circumstance explains the cases in which erythema nodosum appears during the later stage of a tuberculous disease in connection with the measles, whooping-cough, and other illnesses that may produce a passing decrease in allergy; with the ensuing return of allergy, erythema nodosum may sometimes appear.

As erythema nodosum appears at the close of the incubation period of tuberculosis, when the children become sensitive to tuberculin, an attempt should be made in these cases to discover visible manifestations of the tuberculous process, which are to be found in most cases in the form of hilum gland infiltrations, demonstrable by X-rays. Erythema nodosum in children is thus most often a significant symptom suggesting that we are confronted by a recent tuberculous process which must be the object of critical investigation, whether it calls for medical treatment or not. On this point Wallgren expresses the opinion that most children with erythema nodosum, especially before the school age, require careful medical inspection and supervision. It is not the erythema nodosum, but the tuberculous process unmasked by it, that must be treated. The time and nature of the treatment depends on the degree of this affection.

A no less important proceeding which the discovery of a fresh tuberculous process should initiate, is the tracing of the source of infection. In cases where the tuberculous infection first appears as erythema nodosum, the task is in so far facilitated that we can more easily determine the approximate date of infection, for erythema nodosum usually shows itself, as we have mentioned, at the end of the incubation stage, and the incubation period for

tuberculosis is held to be from 3 to 7 weeks. By careful enquiries, dispensary visits in the home, etc., we can succeed in many cases, no doubt, in tracing the source of infection and in taking the necessary steps.

It not rarely happens, however, that infectious consumptives have already caused little endemics of erythema nodosum or tuberculous infections before we have succeeded in tracing them. Such endemics have been described when occurring among children of the same family (Appert[1], Gendron[5], Symes[10], Wallgren[12] and others), among children in a children's home, in hospital wards, among the inhabitants of small towns (Symes, Wiborg[13], etc.), among children in the same building (Belfrage[2]), and in schools (Landau[6], Lundius[7], Wallgren). A number of authors, with the support of such epidemic appearances of erythema nodosum, consider that it is a manifestation of an acute specific infectious disease (Comby[3], Feer[4], Tachau[11], Symes[10]). As against this view Wallgren emphasizes that in all the six family endemics observed by him, as well as in a school endemic, he has after careful investigation found a tuberculous source of infection, and he assumes that the same must be the case with the other epidemics observed. This was also the case both with the house endemic observed by Belfrage, and with the school class infection described by the present writer in 1930. Further carefully investigated cases which throw light on this question may therefore be of interest, and I am able to report of an additional school-class endemic of erythema nodosum observed in Gothenburg.

On March 28th, 1931, a girl of 11 was admitted to the children's hospital from one of the Gothenburg primary schools, for erythema nodosum. In compiling the medical report I made investigations into a possible tuberculous source of infection in the home or among her acquaintances, but none was known. Nor did the patient know of any girl at school who coughed much or had been ill, but when asked point-blank if any other girl in her class had had red spots on the lower extremities of late, she stated that three other girls in the same class had been affected by them at the same time.

All four girls were admitted to the children's hospital, and showed fresh outbreaks of erythema nodosum as well as fresh hilum gland enlargements in the skiagrams.

No suspicious source of infection could be designated, but as there was reason to presume that it could be found in the class, a thorough examination was undertaken of all the members of the class, as well as of the three teachers who did duty there.

The three teachers showed no symptoms of pulmonary tuberculosis, either physically or by X-rays.

The class consisted of 31 girls of 11-12 years of age, who were all tested with tuberculin and examined physically and radiologically. The result was that 29 girls (93·6 per cent.) reacted positively to tuberculin, while only two were negative, even with a Mantoux test up to 3 mgrm. Of the 29 tuberculin-positive girls, one revealed a right-sided cavernous pulmonary tuberculosis, and after repeated tests she showed sparse tuberculous bacilli in her sputum. The general condition of this girl was excellent, free from fever; she was the stoutest in the class, and gave no impression of suffering from active tuberculosis.

Besides the four girls with erythema nodosum (Fig. 1), one of whom afterwards had pleurisy, four of the other girls showed increased sedimentation reaction and by X-rays fresh hilum gland enlargements of such dimensions that all of them were judged to require lengthy hospital treatment. Four more girls had hilum gland

enlargements of uncertain age, the other tuberculin-positive children had probably older infections, four of them showing calcified hilum shadows (Table 1).

TABLE I.

ANALYSIS OF CLASS OF 31 SCHOOLGIRLS INCLUDING 4 CASES OF ERYTHEMA NODOSUM.

Tuberculin reaction.	Disease.	No. of cases.	Per cent.
Positive	Pulmonary tuberculosis	1	93·6
,,	Erythema nodosum	4	
,,	Tuberculous bronchial glands : fresh cases ...	4	
,,	,, ,, ,, uncertain age ...	4	
,,	,, ,, ,, older cases ...	12	
,,	,, ,, ,, with calcified foci	4	
Negative		2	6·7
	TOTAL ...	31	

Discussion.

In a school class of 31 girls of ages from 11 to 12, 93·6 per cent. showed evidence of tuberculous infection, which is, of course, a remarkably high proportion. In the northern countries we can expect not more than 50 per cent. of children of that age in primary schools to be infected. The high figure in the present school-class is explained by the discovery of the girl with pulmonary tuberculosis, who must have been the source of infection to a good proportion of the infected class-mates. This supposition is supported by at least 8 fresh cases of tuberculosis, 4 of which began with erythema nodosum.

Of great interest is the *simultaneous* appearance of erythema nodosum in the 4 girls. In view of the usual appearance of this condition at the close of the incubation time for tuberculosis, there is reason to assume here that the girls had been infected one or two months earlier, and that the infection must have taken place at about the same time in all the cases. There is also a plausible explanation of this, for the form mistress states that the girl with pulmonary tuberculosis, who has not been away from school for a single day all the term, had had a 'cold' for a short period at the end of February, i.e., 4 to 6 weeks before the erythema nodosum cases occurred, and had coughed so much that the teacher had advised her to keep away from school, which the girl had not done. She soon got better, however, and shook off her cough.

In all probability this girl, who has a pulmonary tuberculosis with sparse secretion of bacilli, and who had perhaps for the most part not been dangerous for those around her, contracted on one occasion towards the end of February an acute infection with bronchitis and violent coughing, which had stimulated the excretion of bacilli and made her very infectious. This had resulted in an epidemic of erythema nodosum in four, and of fresh hilum tuberculosis in eight, of her class mates. Whether some of the other tuberculin-positive children had been previously infected by this girl is impossible to decide. That her infectiousness was restricted to a short space of time is supported by the consideration that the two girls who were tuberculin-negative six weeks after this period (when the erythema nodosum appeared), did not react to tuberculin even two months later (after the

FIG. 1. Four of the girls in the class with simultaneous eruptions of erythema nodosum.

source of infection had been removed). It is also to be noted that one of these girls did not join the class until the 5th of March, which may explain her freedom from infection with tuberculosis. If this is so, there remains only one girl out of 29 in the class that had escaped tuberculosis in spite of the possibility of infection from her school-mate.

The present observations not only give further support to the interpretation of erythema nodosum as an early symptom of tuberculosis in children, and throws light on its importance as a diagnostic aid, but also emphasizes the importance of tracing the source of the tuberculous infection, with all its consequences. Thus, in the present instance the presence of erythema nodosum led, after persistent search, to the discovery of an individual with pulmonary tuberculosis, her segregation from an infection-threatened centre (the school), her proper care and treatment, and the

discovery of fresh hilum-gland tuberculosis in eight girls who were consequently let off the strain of school work, to enjoy a much required rest during the first phase of such a serious disease.

In the epidemic of erythema nodosum in one of the primary school classes in 1927, as recorded by Wallgren, all 33 of the girls were exposed to tuberculous infection from a class-mate with pulmonary tuberculosis, and reacted positively to tuberculin; 12 of these developed erythema nodosum and 6 had a period of fever from an unknown cause, all during one and the same term. In 13 of the cases there were hilum-gland affections radiologically demonstrable. The rest had what were probably tuberculous infections of older date.

In 1930 I gave an account of a tuberculous infection in a primary school class at Gothenburg. In a girl of 12, who visited the children's hospital's out-patient department, I found pulmonary tuberculosis with tubercle bacilli in the sputum. In taking down the history of the case I discovered, from questions put to the patient, that 2, possibly 3, of the girls in the class had had erythema nodosum that term, and on a consequent examination of the class I found 25 out of the 30 girls (or 83·4 per cent.) were infected with tuberculosis, and of these, apart from the girls with erythema nodosum, 8 showed fresh hilum-gland affections on X-ray examination.

We may now compare the circumstances surrounding these three epidemics. The numerous cases of erythema nodosum in Wallgren's class led to the whole class being examined, and to the discovery of a girl with pulmonary tuberculosis as the suspected source. In my first epidemic, the discovery of the tuberculous girl induced me to question the patient directly about secondary cases, not only in her home but also at school, with the consequent discovery of the cases in the class. In the epidemic now recorded, a direct inquiry was also made about other cases of erythema nodosum in the class, with a subsequent search for the source of infection, which was only discovered after careful and persistent investigation. None of the girls could be at first sight suspected, and the girl with pulmonary tuberculosis was the last of those examined.

The facts of these three epidemics clearly point to the importance of an exact anamnesis and a search for secondary or primary cases when in the presence of erythema nodosum or tuberculosis in children. They also show that, when dealing with children in the school age, we must not forget to look into the state of things in the class or in the school, on which occasions the staff must not be left out of count should further investigations be pursued.

Another point worth discussing in this connexion is that of the infectiousness of children with bronchial-gland tuberculosis, i.e., not pulmonary tuberculosis proper. Since, by means of irrigation of the stomach and guinea-pig tests, we have now succeeded in finding tubercle bacilli in children with fresh hilum-gland tuberculosis (Opitz[8], V. Poulsen[9],

Wallgren and others), and in some of those with erythema nodosum (Wallgren), this question of the degree of infectiousness is an important one. Proposals have been made to assess the danger of infection according to a certain number of negative tests, but this is an uncertain and capricious proceeding. Theoretically there is, of course, risk of infection, but in practice it is only in a few cases that it has been possible to show proof of a child, with hilum-gland tuberculosis only, having infected another child. According to our experience at the Gothenburg Children's Hospital, we have succeeded in the great majority of cases of hilum tuberculosis in tracing the sources of infection to real cases of pulmonary tuberculosis. The question is, however, of extreme importance, and this has induced me to report at length the circumstances of the three above-mentioned endemics. In all three cases there were sources of infection which had actual pulmonary tuberculosis with cavitation. In the last endemic I thought at first that I should have to be content merely with a suspicion of one of the children with hilum tuberculosis, but finally I found the girl with pulmonary tuberculosis. In the endemics described by me no new infections appeared in the period immediately following the removal of the infectious girl, but I would point out that most of the girls with fresh hilum-gland affections, at the last endemic at least, were segregated at the same time.

I do not cite these cases merely as evidence against the infectiousness of hilum tuberculosis, but to emphasize that in practice, when tracing a source of infection, we should not rest content with a hilum tuberculosis only as the possible cause of other cases of infection.

REFERENCES.

1. Appert. ref. Wallgren (see below).
2. Belfrage, H., *Acta Pædiat.*, Stockholm, (in the press).
3. Comby, *Arch. de méd. d. enf.*, Paris, 1923, XXVI, 329.
4. Feer, *Schweiz. med. Wchnschr.*, Basle, 1926.
5. Gendron, *Bull. et. mém. Soc. méd. des Hôp.*, Paris, 1920, 475.
6. Landau, A., *Münch. med. Wchnschr.*, Munich, 1931, LXXVII, 1663.
7. Lundius, *Acta tuberc. Scandinav.*, Copenhagen, 1929, IV.
8. Opitz, *Münch. med. Wchnschr.*, Munich, 1931, LXXVIII, 949.
9. Poulsen, V., *Jahrb. f. Kinderh.*, Berlin, 1931, CXXX, 127.
10. Symes, J. O., *Erythema nodosum*, Bristol, 1928.
11. Tachau, *Handb. d. Haut-u. Geschlechtkrankh.*, 1928, VI.
12. Wallgren, A., *Handb. d. Kindertuberk.* v. Engel und Pirquet, Leipzig, 1930, 809.
13. Wiborg, *Norsk Mag. f. Lægevidensk.*, Oslo, 1923, LXXXIV, 135.

THE EXTENT OF THE PANCREATIC LESIONS IN DIABETIC CHILDREN

BY

HENRY E. W. ROBERTON, M.A., M.B., B.Ch.

(From Addenbrookes Hospital, Cambridge, and St. Bartholomew's Hospital, London.)

Diabetes in children is usually a progressive disease. After each of the infections from which all children suffer, the insulin requirement becomes permanently higher, even if there is a slight remission during convalescence.

By analogy with nephritis and other progressive diseases, the pancreatic lesions might be expected to become worse until life is no longer possible. However, diabetic children can be kept healthy with insulin even if the disease is of long standing. It is possible, then, that the severe cases may have no power of secreting insulin, or so little as to be clinically negligible.

The patients quoted below were treated on low carbohydrate (e.g., ' Line and Ladder ') diets.

On the whole, their insulin requirements are far above those of most of the diabetic adults treated at the same hospitals.

The C/I ratio.—I have devised a simple method of estimating the insulin-secreting power of the pancreas. It can be used for patients of any age and on any diet of the low carbohydrate type. Though not exact it can be applied to large numbers of cases from the literature or from hospital records. The variations in pancreatic efficiency are large compared with the errors due to the inaccuracies of the method.

The index of severity is the ratio of the potential carbohydrate of the diet to the insulin required to keep the urine sugar-free, in the absence of infections or toxæmia. It is referred to as the C/I ratio.

It is suggested in this paper that when the C/I ratio is between 1·5 and 2 the patient is secreting little, or perhaps no, insulin. It is shown that children tend in time to get the ratio 1·5; after this they remain constant.

The formula used to estimate the potential carbohydrate of the diet is C=0·6P. This is an approximation of the formula[1] C=0·58P+0·1F. For example,

If C=40, P=75, F=100,
Potential C=40 + 0·6 of 75,=85.

Limitations in the use of the C/I ratio.—The diets must be of approximately the same type. If traces of sugar are found in the urine they may be neglected without appreciable error, as was found by sugar estimations on two patients. The insulin must be reduced as far as possible without causing glycosuria, as otherwise unduly low ratios are obtained. The patient must not be in a septic or toxic condition, or passing aceto-acetic acid in the urine.

The method is admittedly inexact, but the results are sufficiently constant to justify its use.

Estimations on cases in the literature.—In Toverud's series[2] the children tend in time to require a constant 28 to 56 units, after 2 or 3 years.

As, unfortunately, only the insulin requirement is given, and not the diet, except in the case reports, the decreasing ratios cannot be demonstrated.

Some of the milder cases after 4 years still have a high ratio. The fact that the 3-year group has higher ratios than the later groups is difficult to explain. The most likely explanation is that the severe cases died early in the disease in the earlier years.

1923 series:—4 years duration, of 8, 4 died (50 per cent.).
Ratios of the survivors, 1·3, 1·3, 1·7, 2·3.

1924 series:—3 years duration, of 23, 11 died (47 per cent.).
Ratios of the survivors, 1·7, 2·1, 2·1, 2·2, 2·2, 2·8, 2·9, 3·1, 3·4, 3·5, 4·0, 4·7.

1925 series:—Of 10, 2 died (20 per cent.).
Ratios of the survivors, 1·0, 1·4, 1·7, 1·7, 1·9, 2·5, 2·7, 3·8.

The figures were obtained in 1927. The diets were of the order of C.P.F.=1.1.2-3.

Joslin[3] gives the food and insulin requirements in 1928 of 11 cases of long duration. He only gives the diets of two in full, of the rest he gives the carbohydrate. C/I ratios have been worked out on the assumption that the other diets were similar. The error is unlikely to be more than 10 per cent.

Duration of disease 9 years, C/I ratios 6·0, 3·4, 1·9.
,, ,, 8 ,, ,, ,, 4, 2·5.
,, ,, 7 ,, ,, ,, 4, 2·8.
,, ,, 6 ,, ,, ,, 1·9, 1·8, 1·7.
,, ,, 5 ,, ,, ,, 2·6.

I have collected a series of cases from Addenbrookes Hospital, Cambridge, up to 1931—

Duration of disease 8 years, C/I ratios 1·6.
,, ,, 6 ,, ,, ,, 3, 1·1.
,, ,, 5 ,, ,, ,, 3·1, 2·4, 1·5.
,, ,, 4 ,, ,, ,, 4·3, 1·3.
,, ,, 3 ,, ,, ,, 1·7.
,, ,, 2 ,, ,, ,, 4·5, 2·9.
,, ,, 1 ,, ,, ,, 3·3.

And a further series from St. Bartholomew's Hospital (up to 1931, unselected)—

Duration of disease	8	years,	C/I ratios	2·7,	1·8.				
,,	,,	7	,,	,,	,,	4,	3,	3,	2·3.
,,	,,	6	,,	,,	,,	1·4,	1·4.		
,,	,,	5	,,	,,	,,	4,	3·6,	2·2,	1·3.
,,	,,	4	,,	,,	,,	3·6.			
,,	,,	3	,,	,,	,,	2·4,	1·2.		
,,	,,	2	,,	,,	,,	2·2,	1·3.		
,,	,, under	2	,,	,,	,,	5,	3·5.	3,	2·3.

It is interesting to note that adults recovering from coma or ketosis frequently have a C/I ratio of about 1·5 for a few days. After this they return to their usual 3 or 4. The coma may be largely due to the toxæmia temporarily putting the pancreatic secretion out of action. In the early stages of recovery, though the disease is controlled by insulin, the pancreas has not started to secrete properly. Such cases seldom have ratios below 1·5 after ketosis has been stopped.

I have not yet succeeded in finding more than one case whose ratio, when stabilized, is below 1. This case (Lawrence[6]) was not typical diabetes, and there were other factors than pancreatic deficiency.

The glucose equivalent of insulin.—Woodyatt[4] found that the unit of insulin was equivalent to 1—1·5 grm. glucose. He put patients on an extremely low carbohydrate diet (on the verge of ketosis) with a great excess of fat, and estimated the daily output of sugar, which was considerable. With a constant diet the same patient passed 1—1·5 grm. less sugar for each unit of insulin.

Allen[5] found that in depancreatized dogs the unit was equivalent to 3—7 grm. sugar. They were on a high carbohydrate diet, about 100—150 grm., with 20—30 grm. protein, and no fat other than that contained in lean meat. He allowed for some of the protein being burned as carbohydrate. The dogs had a considerable glycosuria. The lower the output of sugar, the lower the sugar equivalent of insulin.

The patients described in my series are not strictly comparable to the diabetics and dogs mentioned above, as they were sugar free or nearly so, and were on diets between the low carbohydrate of Woodyatt's patients and the high carbohydrate and low fat of the dogs.

Had Allen's dogs and Woodyatt's humans been sugar free, the latter also always ketone free, the values would have been slightly different, probably a lower C/I value.

Whereas I estimated my patients' total sugar and insulin, in these experiments only alterations of sugar in response to insulin were estimated. Calculations of the C/I ratio on these patients were impossible on account of the gross glycosuria.

The unit of insulin appears to correspond to 1—1·5 grm. glucose on a low carbohydrate diet, and to 3—7 grm. on a high carbohydrate diet.

A total diabetic with a diet of the order of C.45, P.74, F.130 would therefore be expected to have a ratio between these extremes. When a patient is changed from a diet of this type to a diet of C.150, P.50, F.50, the C/I ratio rises.

The view that these children are total diabetics is therefore consistent with the results of these experiments.

Survey of the C/I ratios of all the available children.—The tendency of the C/I ratio to fall to 1·5 is shown in two charts.

Chart I.—The curve shows the number of children with a given ratio, in groups of 0·5. The greatest number of cases is in the group 1·5. There are few below this. There must be some factor preventing the pancreas from degenerating further. The cases are taken from St. Bartholomew's Hospital, Addenbrookes Hospital, Toverud, and Joslin.

CHART I.

C/1 RATIO LAST RECORDED, OF 65 CHILDREN.
INCIDENCE OF C/I RATIOS IN GROUPS OF ·5.
NONE BELOW 1. MAXIMUM AT 1·5.

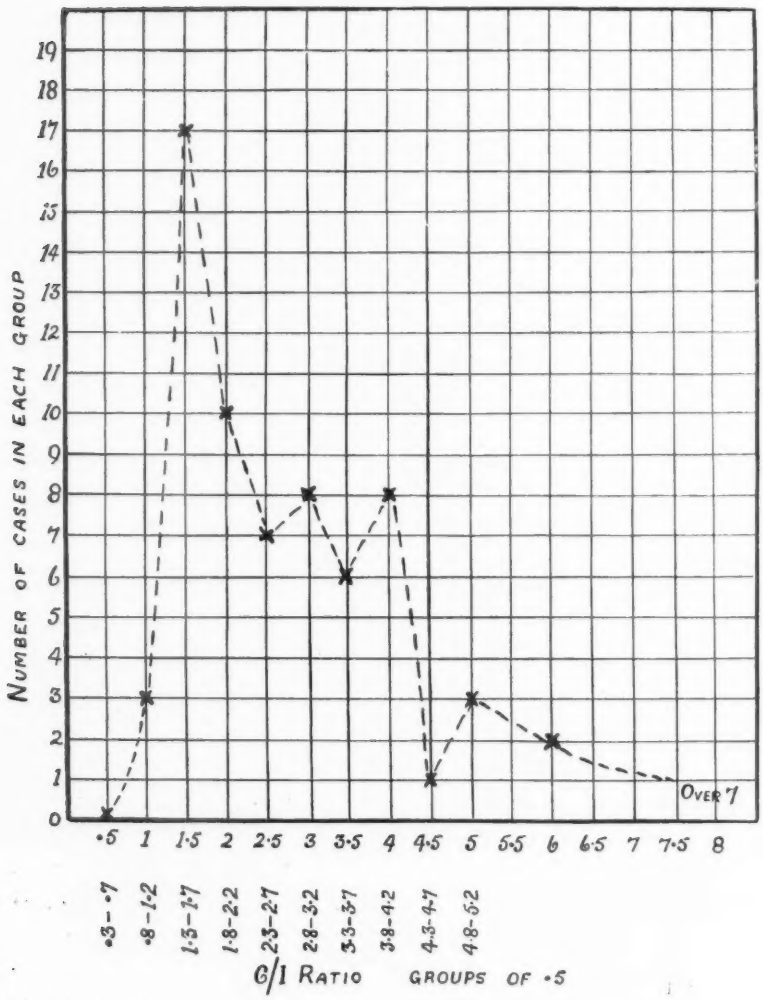
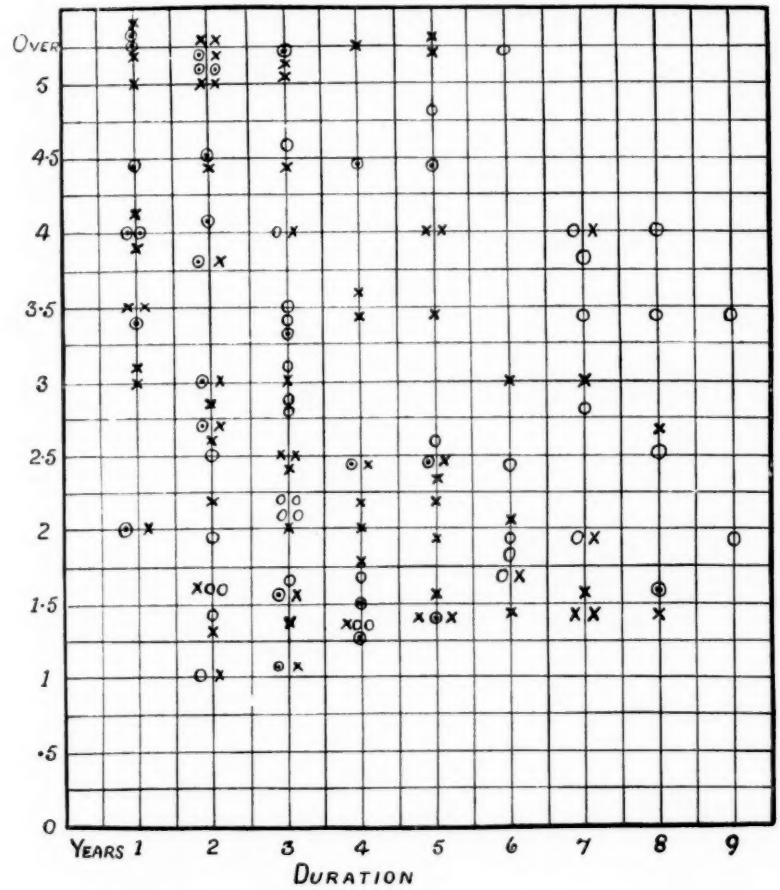


Chart II.—The chart shows that a few cases remain mild. The C/I ratio is plotted against the time since diabetes was diagnosed.

CHART II.

Chart of C/I ratios and duration.



X = St. Bartholomews ⊙ = Addenbrookes O = Toverud & Joslin

Summary.

During the last few years children with diabetes have tended to require progressively more insulin until they require 1 unit for each 1·5 grm. of potential carbohydrate in the diet. It is suggested that at this stage they are not secreting an appreciable amount of insulin, because (a) experiments show that 1 unit corresponds to about 1·5 grm. of glucose; and (b) exhaustion of the pancreas is the simplest explanation of the disease ceasing to be progressive at this stage.

My thanks are due to Dr. George Graham and the physicians of Addenbrookes Hospital for permission to use their cases and clinical records.

REFERENCES.

1. Woodyatt, R. T., *Arch. Int. Med.*, Chicago, 1921, XXVIII, 125.
2. Toverud, K. U., *Brit. J. Child. Dis.*, Lond., 1927, XXIV, 258.
3. Joslin, E. P., *Treatment of Diabetes Mell.*, Philad., 1928, 4th Ed.
4. Woodyatt, R. T., *J. Metab. Res.*, N.J., 1922, II, 795.
5. Allen, F. M., *Am. J. Physiol.*, Baltimore, 1924, LXVII, 275.
6. Lawrence, R. D., *Quart. J. Med.*, Oxford, 1928, XXI, 359.

DIFFICULT AND DEFECTIVE CHILDREN

BY

A. DINGWALL FORDYCE, M.D., F.R.C.P.,

Physician, Royal Liverpool Children's Hospital;

Certifying Officer, Liverpool Education Authority.

A child whose management and training never present difficulties is not a normal child, and when we speak of a ' difficult ' child, we mean by the term special difficulty either in degree or nature, or in both. The difficulty is frequently one of behaviour, such as, for example, disobedience, lying, fighting, temper, thieving, enuresis. All such are instances of difficulty of excessive degree, because the normal child is occasionally disobedient, lies, fights, has fits of temper, makes petty thefts, and wets himself or his bed. But the difficulty may be different in nature. It may be of a social nature and show itself at home or at school in bullying, wanton hurting, apparently causeless turbulence, and possibly wandering away; or of a physical symptomatology such as sleeplessness, twitchings, refusal of food, indigestion and anæmia.

Difficult children.

Causative factors.—Confronted with a difficult child we have to consider primarily the cause of the trouble, and naturally we turn first to examination of the child himself. As the underlying cause of difficulty in the cases of normal children is often temporary physical disability, we seek first a physical cause, and among such causes and often unobtrusive in character are throat infections, bacilluria, fatigue and indigestion.

Indigestion may be either the cause or the result of difficulty. When it is the cause, its successful treatment cures the difficulty: when it is the result, medicinal and dietetic treatment are naturally inefficacious, and further investigation and action are indicated.

Not only the physical condition, but the mental state also demands examination. Is the child mentally defective, or although mentally within the limits of the normal, does he possess innate temperamental qualities which render specially directed management advisable? Many mentally defective children are difficult children but there are many difficult children who are mentally normal, and the responsibility for the cause of the difficulty may not rest with the child at all but be environmental in nature, and rest mainly on the shoulders of his parents or guardian. Guardianship may be unwise or irresponsible; parental care may be foolish or faulty; there may be lack of supervision or excessive care or mal-administration.

The doctor in the past has generally been regarded as a physical adviser and much of his work still is purely such, but he must also be to some extent a psychiatrist. He must, when necessary, probe into mental and temperamental conditions, personal, domestic and scholastic, and be prepared to give advice and act as conditions demand. Difficulties, ungrappled with, may lead to serious disorder—physical, mental and social.

When the fault lies solely with the child we may be able to do a great deal by restoring normal health, and when parents or guardian are willing and able to aid us, much can be done when the source of difficulty is mainly environmental. Unfortunately, however, only too often we are not allowed, or have not the opportunity, seriously to deal with the problems which exist. All of us with families know that it is supremely hard to be a good parent, even without reading the parents' questionnaire set by our American friends. The neurotic or psychopathic parent resents criticism, discredits explanation, and flits with her child from doctor to doctor. The two alternative courses which can be followed are either to place the facts of the position plainly before the parent and attempt to obtain the desired change in conditions by the force of reasoning, or so to deal with family affairs, including possibly a holiday for the mother, as to obtain the conditions necessary for improvement of the child.

Perhaps the most difficult cases are those in which disharmony exists between mother and father. If parents are divorced or separated, not only is the enormous value of biparental influence and care lost, but parental influence is not infrequently so perverted as to be actually harmful.

It is common to find that the difficult child is of neurotic heredity. The spoilt child is frequently the result of an inferiority complex on the part of the mother, or irresponsible indulgence on the part of one or other parent. Sometimes difficulty is due to excessive firmness in control, either on the part of parents or of school teachers. The error of the parents in such cases may be due to lack of sympathetic discernment, or at other times to misguided or excessive zeal for the welfare of their child. When the difficulty is the result of school control, its basis is often an unrecognized physical disability, frequently one of the special senses.

Excessive and misguided care by parents is part of a modern turn towards the psychological aspects of health and illness, and only too often serves as an illustration of what has been termed in the States the 'menace of psychiatry.' Thus we meet with mothers intensely aware of the power they wield in fashioning the health and happiness of their children through psychical influence. Such mothers, awed by this knowledge, may on the one hand flutter ineptly, and by their vacillation merely encourage the development of childish disorder; or on the other hand, obstinately resolute in carrying out their superficial psychiatric information, they may cause trouble through misguided effort.

We have experienced the power and also the danger of the results of the psychological states of doubt, indecision, and fear in relation to the diet and the digestion of young children, and particularly babies. A doubting,

undecided, fearful mother gives her baby indigestion, whether it be breast- or bottle-fed. The lay parent, inflated with hearsay knowledge of all the vitamins, and all the superficial bio-chemistry of the food-stuffs, runs a danger of continuing the trouble. In like manner there is a danger to children in the unwise exercise of lay and domestic psychiatry. This danger is fostered by the widespread use of standard psycho-metric tests, the development of Child Guidance Clinics, the free discussion of the definition of mental defect and the limits of certification, and the increase of attention to feeble-minded children. All such activities are unquestionably useful and desirable, but require to be kept in proper perspective, and our difficulty is in preventing the lay individual from applying tests, observations, supervision, etc., which while true and correct generally for the mass, are only of value in the case of the individual child when used with suitable discrimination, and when considered along with all other factors.

Remedial measures.—During the past 50 years, and largely owing to the passing of the several Education Acts, there has ensued a gradual tabulation of groups of children with an educational object in view, but largely on medical lines. Medicine and education have become more and more closely intertwined until to-day in our various Special Schools, Hospitals with Schools, and Residential Institutions of different kinds, the two are conjoined in one. With the division of children into massive groups, there has been separated as a special group those children who are described as ' maladjusted,' ' nervous,' ' difficult ' ' unmanageable,' or ' neurotic,' and to deal with the problems their cases present Child Guidance Clinics have been instituted. Some of these children are a-social, some are unable to fit into their environment, and in many cases the environment is such as to prohibit normal development.

It is not only or indeed mainly Child Guidance which is called for, rather is it the guidance or manipulation of environmental conditions to fit the needs of the child.

To carry out the work, the clinic staff is based on the investigations of a social worker, a lay psychologist and a medical psychiatrist working in the closest union and in association with all authorities and services dealing with children. The mental and physical state of the child is determined, all available information regarding home, school, etc., is obtained, the cause or causes of difficulty are probed and decided upon, and a suitable form of remedy or remedies is formulated and efforts made to have it carried out. These children may be physically healthy or physically defective, and they may be mentally normal or mentally defective.

If they are physically defective, measures are taken to ensure proper treatment, and if they are mentally defective they are referred for certification and consequent suitable disposition.

A Child Guidance Clinic properly staffed does good work which otherwise will not be carried out. This type of Clinic is certain to develop; and though it is possible and, I think, right to regard it as only a temporary necessity, yet

until medical and social conditions develop considerably, it is bound to fill a gap in prophylactic and therapeutic pædiatrics. Its work does not encroach on that of the general practitioner, and further the work is such as cannot at present be done by the general practitioner. Really good work at a clinic can only be carried out when the controlling head is a pædiatrist with psychiatric training, and pædiatrics in this country is still regarded as so subsidiary a subject in the undergraduate curriculum that only the experienced general practitioner can be, as so often he is, a valuable adviser in the subject. The work is most time-absorbing, and until society recognizes the need and is able and prepared to recompense doctors adequately for work performed, it is impossible for the individual to undertake it.

Defective children.

Just as ' difficult ' is a vague term, so likewise is ' defective,' and no exact medical meaning is attached to the expression. We all constantly meet with defective children of many types, and in private practice, if we use the adjective at all, we may employ it loosely and as indefinitely as we please.

Classification.—But different types of defective children are defined by Acts of Parliament, the main Acts concerned with the subject being the Education Act, 1921, and the Mental Deficiency Acts, 1913-1927.

The definitions in these Acts were formulated for specific purposes, education, segregation, protection, etc., and in their construction and declared signification pass beyond pure medicine. It is therefore possible to attach different meanings to the terms, according as they are employed in the official sense or otherwise; or conversely, to have differing opinions on the proper designation of a particular child. In the formal use of these terms, however, it seems only wise and right to employ them in the official sense, and particularly is this the case when dealing with mental defect. Accurate diagnosis not infrequently discloses double defect, physical and mental.

In all cases both medical care and education are important factors in treatment, and it is imperative to decide whether the calls of medical care should be paramount with education as subsidiary (e.g., prevention of established rheumatic carditis, cure of tuberculous joint), or whether the outstanding factor in placement is educational and depending on mental capacity (e.g., birth trauma).

I have already spoken of the ' difficult ' child, and the ' difficult ' child may be and often is regarded as a bad child. No wise man can minimize the value of a good mental and moral heredity, or fail to recognize the success that usually accrues when this is conjoined with a good mental environment. On the other hand a child of poor heredity with correspondingly poor mental environment may be naturally inclined to be naughty or bad: we must, in a

word, accept the existence of the bad child while emphasizing the causal importance of environment.

There is also, defined by statute, the child who is to be termed a ' moral defective,' i.e., the child ' in whose case there exists mental defectiveness coupled with strongly vicious or criminal propensities and who requires care, supervision and control for the protection of others.'

The primary essential, then, in concluding that the misdeeds of a child are such as to bring it within this classification, is that he should be considered mentally defective. If his badness is to be regarded as the evidence of mental defect it must be demonstrated that he does not and cannot recognize the difference between right and wrong, or else we must conclude that there exist general or other grounds for considering him mentally defective and that his badness is part of general mental lack.

Feeble-minded children of both sexes are often easily led into evil ways, but a true moral defective is uncommon in childhood. During the six years, 1925 to 1930 inclusive, there were certified as moral defectives seven children of school age in Liverpool, five of these being post-encephalitics. Fifteen other children certified as feeble-minded were excluded from school because of conduct detrimental to other children.

So far as the special senses are concerned, the interpretation of the terms ' blind ' and ' deaf ' in the Act (Ed. Act, 1921, p. 42) is as follows:—The expression ' blind ' means too blind to be able to read the ordinary school books used by children: the expression ' deaf ' means too deaf to be taught in a class of hearing children in an elementary school.

Certification.—A child is certifiable as physically defective when ' by reason of physical defect he is incapable of receiving proper benefit from the instruction in the ordinary public elementary schools, but is not incapable by reason of that defect of receiving benefit from instruction in special classes or schools.' (Education Act, 1921, p. 35.)

Under this certification many surgical cases, mainly orthopædic, obtain medical supervision and education both in residential and day special schools. While there is much difference of opinion regarding the desirable placement of many cases after they have ceased to require residential care, the special practical training, and the special facilities for continuous surgical supervision in day special schools are important considerations. In these day special schools also many purely medical cases, such as of severe heart disease, derive benefit unobtainable in their cases in a public elementary school.

The subject of mental deficiency is more complicated than that of physical deficiency, the primary difficulty being in the precise interpretation of the definition. Many misconceptions exist regarding it both among the laity and members of our profession, and it is regrettable when the term is professionally employed in obvious ignorance of the signification attached to it. Of the classes of persons who are to be deemed mentally defective apart from moral defectives, those of the lowest grade are idiots, those of the next grade imbeciles, and the highest grade of all are feeble-minded.

Idiots and imbeciles are from the purely medical aspect unmistakably mentally defective children, a good example of the latter being a mongol. Feeble-minded children sometimes are clearly defective, but very often accurate diagnosis demands intimate personal knowledge, as well as reports from various sources. Among the higher-grade feeble-minded, the border-line cases, a probation period for special observation is usually essential.

Feeble-minded children are defined in the Mental Deficiency Act as persons ' in whose cases there exists mental defectiveness which, though not amounting to imbecility, is yet so pronounced that they appear to be permanently incapable, by reason of such defectiveness, of receiving proper benefit from the instruction in ordinary schools.' This definition, on the face of it, appears purely scholastic, and this aspect is endorsed by the section in the Education Act, 1921, dealing with the subject, where feeble-minded children are considered those who by reason of mental defect are incapable of receiving proper benefit from the instruction in ordinary schools, but are not incapable of benefiting from instruction in special schools.

The response to scholastic environment and instruction is considered in both Acts a just criterion of social adaptability and potentiality; or, to express this from the practical point of view, school status and progress, regarded from a broad social aspect, is the most important test of a child's mental capacity.

It seems right that such a concept should form the basis of diagnosis, and if advisable certification; and in 1929 a Committee, appointed by Sir George Newman to consider the problems presented by the mentally defective child, reported that in its opinion mental defect should be regarded as a lack in capacity of social adaptation, either primary or secondary in origin.

Certification of mental defect is governed by the definition in the Mental Deficiency Act, 1913, as amended in 1927. Here it is laid down " for the purposes of this section ' mental defectiveness ' means a condition of arrested or incomplete development of mind existing before the age of eighteen years, whether arising from inherent causes or induced by disease or injury." This gives very wide latitude in diagnosis of the fundamental condition, particularly as the onset of mental defectiveness is noted as being possible at any time up to the age of eighteen years.

Many early cases of secondary amentia do not live till school age, and these cases of early death are mainly in the lower grades of mental defect. Some cases of mental defect are not obvious before school age, and these are usually of the higher grade and primary in nature. Thus at school age primary amentia is much more common than secondary amentia, and a great deal of it is of high-grade, that is, among the feeble-minded.

The fact that by statute mental defect is recognized as originating at any time up to the eighteenth year of life, necessarily modifies this statement, but it remains true that the great majority of high-grade mental defectives of school age are cases of primary amentia.

During the seven years 1925 to 1931 inclusive, there have been certified in Liverpool 2,128 children of school age as mentally defective—namely, 1,540 feeble-minded, 530 imbecile, 32 idiot, and 26 detrimental to other scholars.

About one-third or 30 per cent. of cases which are referred for examination as suspect are after a period of probation permanently retained in the ordinary school; no child is certified mentally defective until it appears conclusively proved that he comes definitely within the scope of the terms of the Act.

The fullest reports from head-teacher, school medical officer, and any agency interested in a particular case, are in the hands of the certifying officer—a full series of intelligence tests is made, personal examination carried out, and frequently a period of probation in an ordinary school is granted.

The numbers given are out of a school population of approximately 140,000, or about 250,000 school children in seven years. They show then that while about one in 85 school children have been reported as suspect of mental defect to the certifying officer, about one in 130 have been certified as mentally defective; and that about one in 10,000 school children have been certified and excluded school because of their behaviour.

In the Special Day Schools for feeble-minded children, the desirable intelligence quotient at entrance appears to be between 60 per cent. and 70 per cent. A good scholastic standard for the leaving child of 16 years is that equivalent to the normal child of 9 or 10 years, and most of these children are fully competent to deal with a humble job.

There are certain basal problems still undecided. Thus it is generally recognized that for mentally and scholastically retarded children, special educational attention is necessary; but there is no unanimity of opinion as to either the wisest definition and separation of groups within the limit of retarded children, or where the special attention necessary for different groups should be obtained.

The basal medical problem is sometimes insoluble:—is this borderline child retarded mentally and scholastically because of inherent mental defect, or because of lack of opportunity, of proper care, of illnesses or injury?

The position is greatly simplified by statute which calls for no exact differentiation in ætiology; but such broad generalization in classification certainly justifies the question whether or no the term mental defective is wise or right for higher-grade borderline cases in childhood.

In considering the figures given above for the years mentioned it has to be borne in mind that for the previous years—particularly the immediately preceding nine years of school life—ascertainment was inevitably less systematic and thorough, and consequently there is certainly included in them an unknown number of certified children carried over from a previous time. The term ' mentally defective ' still carries with it a stigma, and

is one which may automatically cease to be applicable to the individual on reaching the age of 16 years. At this age certification in terms applicable to childhood ceases, and unless further steps are taken the mentally defective child does not proceed to become a mentally defective adult.

Of 1,200 children certified as feeble-minded in the last six years, 170 automatically became decertified on reaching the age of 16 years (roughly 15 per cent.), while 93 were decertified before that age (roughly 8 per cent.), i.e., in all about 23 per cent. of those children certified as ' feeble-minded ' were not certifiable at the age of 16.

I would quote a recommendation from the Report of the Mental Deficiency Committee of the Board of Education and Board of Control, 1929 (p. 157), in which it is stated: ' We recommend the abolition of the requirement that the Local Education Authority should certify a particular type of child as mentally defective as a necessary preliminary to providing him with the type of education he requires.'

FOUR CASES OF URINARY OBSTRUCTION

BY

A. V. NEALE, M.D., M.R.C.P.

(From the Children's Hospital, Birmingham.)

The frequency and importance of urinary tract defects, obstructions and infections in children is now well recognized, but unfortunately the recognition is often delayed until an emergency state of severe obstruction, uræmia or superadded infection brings the case under observation.

In a very interesting and comprehensive paper by Poynton and Sheldon[1], the causes and effects of dilatation of the bladder and ureters in children are outlined and there is very little new information to be added to their observations. Therefore, the only reason for placing the following cases on record is in order to extend further the clinical illustrations of these particular disorders. Four cases will be outlined, in each of which a definite organic cause was found to be associated with dilatation of the bladder, ureters and renal pelves, all instances occurring in male children.

The cases here presented showed the cause of the disorder to be situated in the posterior part of the urethra, and in each instance a well recognized pathological state was demonstrable.

The following cases are presented:—

Case 1. Congenital urethral valves.
Case 2. Congenital urethral stricture.
Case 3. Sarcoma of the base of the bladder.
Case 4. Urethro-vesical calculus.

Case reports.

Case 1. Congenital urethral valves (with Figure 1).

Baby G., aged 9 days, male; third child in healthy family. At birth it was noticed that the recti muscles of the abdomen were widely separated, and later the umbilicus was observed to be weeping.

The baby was brought to hospital in a very poor physical condition, the mother stating that the child had passed very little urine since birth, and that there was unusual swelling of the abdomen. The bowel had acted normally, but feeding had been difficult. It was obvious on admission to the hospital, that the urinary bladder was very considerably distended and extremely tense. Careful abdominal palpation also revealed the outlines of distended thickened ureters and enlarged lobulated renal swellings.

A clinical diagnosis of congenital urethral valves was made. Very small quantities of urine escaped from the urethra, but the introduction of a small rubber catheter presented no difficulty and vesical drainage was established. There was no sepsis in the urinary tract and the urine was normal. The infant failed to survive.

Autopsy. At the necropsy an excellent example of urinary obstruction due

to the presence of congenital mucosal valves in the posterior urethra was found, an arrangement whereby pressure by fluid in the vesical side caused closure of the posterior urethral canal. The drawing (Fig. 1) illustrates the extreme vesical hypertrophy and urethral dilatation. On the right side of the prostatic area a small acute abscess was present, this being apparently due to direct infection in the prostatic ducts, and being so placed as to cause quite complete occlusion, by lateral pressure, of the prostatic urethra.

There was no evidence of any other congenital physical defects in the child.

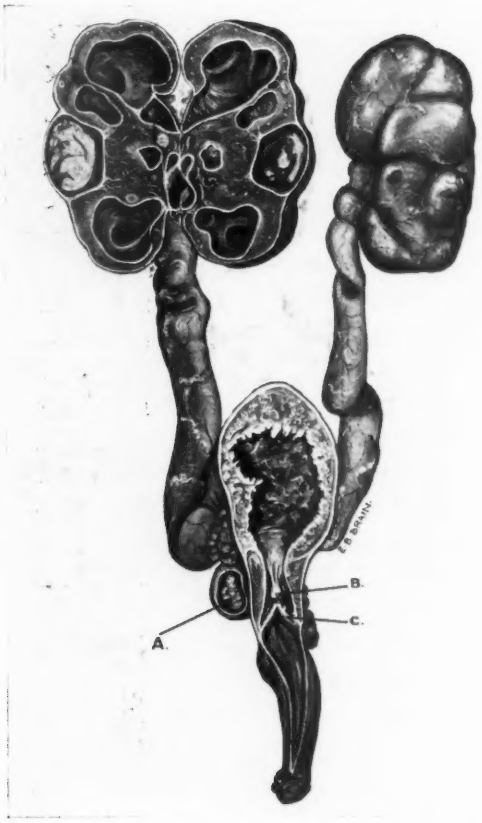


FIG. 1.—Case 1, Baby G., aged 9 days; showing congenital urethral valves.

A. Perivesical abscess.
B. Prominent verumontanum.
C. Urethral valves.

Case 2. Congenital urethral stricture (with Figure 2).

R. C., aged $4\frac{1}{4}$ years, male; first of four children, others healthy. The child was 10 lb. at birth and made good progress until pyuria was first discovered at about two years of age. This urinary infection never improved materially.

At the age of $4\frac{1}{4}$ years this child was first seen at the Children's Hospital with a recent history of melæna. The general physical condition was moderate and there was no evidence of cardio-vascular disease, but the respiration was uræmic in type and the urine showed a heavy pyuria and infection with bacillus coli and streptococci. The bladder was observed to be somewhat dilated continuously, but urine was evacuated at intervals. The pyuria failed to respond to medicinal therapy and progressive uræmic phenomena, including vomiting and severe hæmorrhagic entero-colitis supervened.

Autopsy. Necropsy revealed slight hypertrophy of the left ventricle without any evidence of intrinsic cardio-vascular disease. Each kidney was small and consisted of sacs containing thick purulent exudate. Both ureters were very distended, tortuous and thickened (1 in. diameter). The bladder was distended and somewhat thickened. The uretero-vesical orifices were dilated. The seat of urinary obstruction was found to be in the posterior urethra where, as shown in the

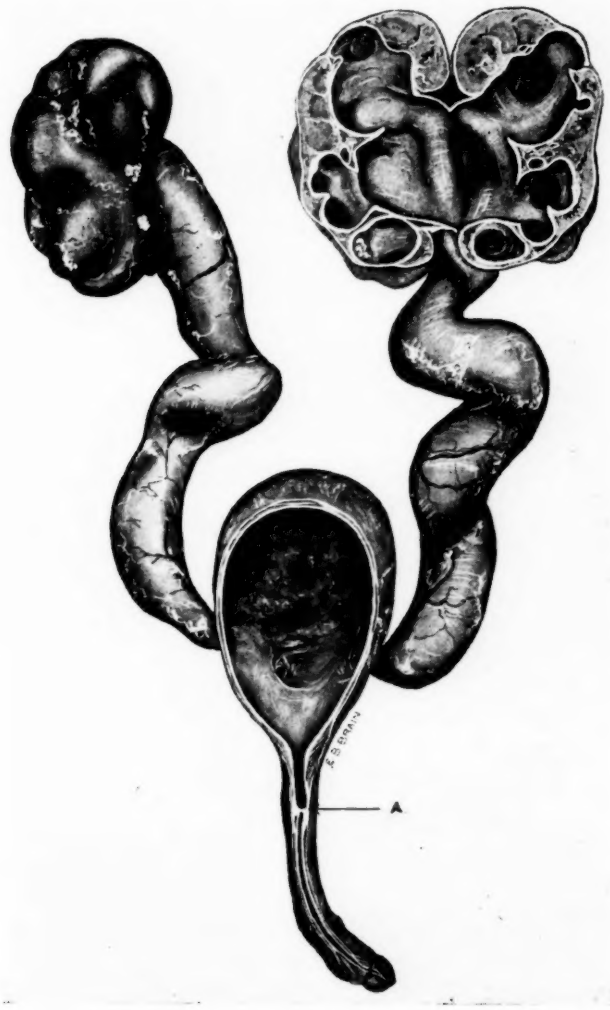


Fig. 2.—Case 2, R. C., aged 4½ years, showing congenital urethral stricture.

A. Stricture.

drawing (Fig. 2), congenital narrowing of the urethral channel was present, and a small fold of thickened mucosa was also seen at this level. A narrow channel through the stricture could be determined, but apparently the associated renal tract infection had caused added mucosal swelling in the stricture area, thus rendering the urethral passage extremely narrow. The urethra distal to the stricture and the meatus urinarius were normal.

Case 3. Sarcoma of base of bladder (with Figures 3, 4 and 5).

C. L., aged 1 year and 2 months, male; third of three children. Breast fed for nine months and made very good progress.

The first indication of urinary tract disease was occasional dysuria, and the detection of pyuria at the age of one year. The urinary tract infection failed to respond to medicinal therapy, and at 14 months of age the child was admitted to hospital with urinary retention; the bladder being very considerably distended and

FIG. 3.—Case 3, showing sarcoma of the base of the bladder.

presenting as a hard suprapubic tumour. Rectal examination revealed a large mass disposed around the base of the bladder, the nature of which at this time was somewhat uncertain. My surgical colleague, Mr. Stammers, attempted to pass a small bougie along the urethra into the bladder without success, due to an obstruction near the posterior part of the urethra and neck of the bladder. Suprapubic cystotomy was performed and urinary tract drainage established, so producing a considerable temporary improvement in the child's general appearance. A few days later a smooth elastic mass was palpable in the perineum. Within ten days of this

observation the perineal swelling had reached at least four times the size noticed before, had become hard and come to occupy practically the whole of the true pelvic cavity. There was no doubt that this was a rapidly proliferating neoplastic mass arising in the region of the prostatic gland.

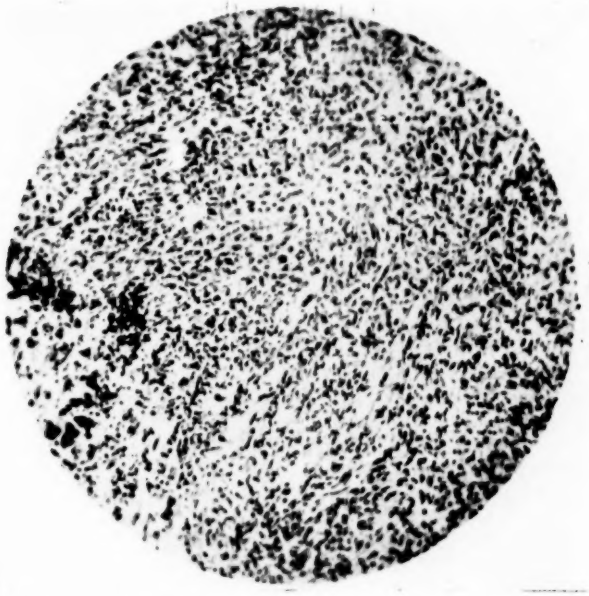


FIG. 4.—Case 3, showing sarcoma of the base of the bladder (low power).

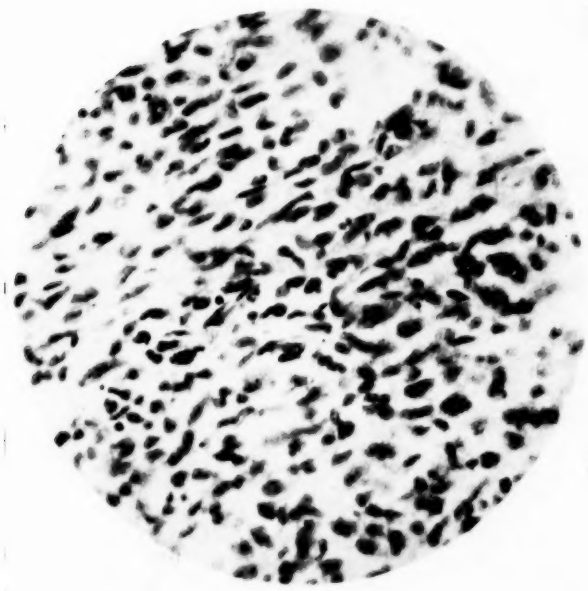


FIG. 5.—Case 3, showing sarcoma of the base of the bladder (high power).

AUTOPSY. At post-mortem examination the pelvic cavity was chiefly occupied by a soft neoplastic mass arising from the neck of the bladder and diffusely infiltrating

the perineum and the ileo-psoas muscles. The wall of the bladder was grossly thickened and both ureters much dilated. The renal pelves were similarly distended with moderate compression, chiefly of the renal substance. Careful examination appeared to show that the neoplasm arose in the anterior part of the prostate gland, but owing to the diffuse nature of the infiltration certainty on this point was not possible. There were no metastases. The drawing (Fig. 3) illustrates the extent and site of the tumour and its relationship to the urinary tract obstruction.

Microscopical examination showed the neoplasm to be sarcomatous in type, and the microphotographs show the detailed histological structure (Figs. 4 and 5).

Case 4. Urethro-vesical calculus (with Figure 6).

J. B., aged 14 months, male. The child was apparently quite well until three

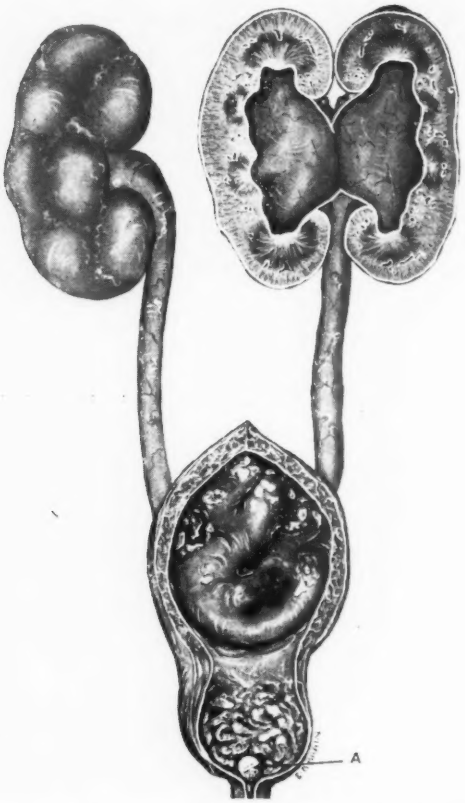


FIG. 6.—Case 4, showing urethro-vesical calculus.
A. Calculus

weeks before admission to hospital. Some difficulty, however, in micturition had occurred from time to time, and circumcision had given no improvement. Severe straining at micturition, and only small amounts of urine had been passed just prior to admission.

Observation showed the child to be pale, drowsy, sometimes almost comatose, with contracted pupils and uræmic deep respiration. A large distended hard bladder was present. A soft catheter was introduced with considerable difficulty and a small amount of thick purulent urine escaped. Suprapubic cystotomy was performed under local anæsthetic (Mr. Stammers), and masses of membraneous necrotic material found and removed from the neck of the bladder. No calculus was detected at this time. Broncho-pneumonia supervened.

Autopsy. Each kidney was enlarged and showed diffuse pyelonephritis, with dilatation of the ureters. Dissection revealed at the neck of the bladder and posterior urethra, a soft mass of collagenous material consisting of necrotic mucosa and fibrin, in the centre of which was an irregular white calculus the size of a small bean, so placed as to cause almost complete urethral obstruction. The drawing shows the existent condition (Fig. 6).

Comments.

The four cases here recorded and illustrated afford good examples of the particular disorders found.

In the case of congenital urethral valves (Case 1), it is unusual for urinary obstruction to occur in an infant so young as in the instance here noted. In this patient the urethral mucosal folds were unusually well marked and the valvular effect was considerable. In cases which are detected before gross urinary obstruction and sepsis have occurred satisfactory treatment has been evolved, but unfortunately such added difficulties are frequently present when the patient comes under observation.

Congenital urethral stricture (Case 2) is apparently an extremely unusual cause of urinary obstruction in young children, although strictural narrowing of a ureter is among the commoner forms of congenital abnormality in the urinary tract.

Chronic urinary sepsis as a sign of disease in the renal tract requiring careful and extended investigation is again indicated.

I am indebted to Professor Parsons, Dr. Smellie and Dr. Gittins for co-operation in obtaining the clinical and pathological notes, and to Mr. Brain for the drawings.

REFERENCE.

1. Poynton, F. J., & Sheldon, W., *Arch. Dis. Childh.*, Lond., 1927, II, 251.